VOLUME 8 • JANUARY 1950 • NUMBER 1

Journal of ORAL SURGERY

AMERICAN DENTAL ASSOCIATION

VOL. 8 • JANUARY 1950 • NO. 1

A JOURNAL

DEVOTED TO THE ART

AND SCIENCE OF

ORAL SURGERY

JOURNAL OF ORAL SURGERY

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Journal of Oral Surgery, Vol. 8, No. 1, January, 1950. Published by the American Dental Association, 222 East Superior St., Chicago 11, Ill. Yearly subscription $5.00. Single copy $1.50. Entered as second class matter June 21, 1943, at the post office at Chicago, Ill., under the Act of March 3, 1879. Published quarterly. Copyright 1950 by the American Dental Association. The Journal of Oral Surgery is indexed in Index to Dental Literature and Quarterly Cumulative Index Medicus.

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BERTO A. OLSON

BERTO ALEXANDER OLSON was elected president of the American Society of Oral Surgeons on October 17 at the thirty-first annual meeting in San Francisco. Dr. Olson was born and lived the greater part of his youth in Buxton, North Dakota. He attended St. Thomas College, St. Paul, Minnesota, and the University of North Dakota, Grand Forks. He left the University of North Dakota to join the United States Army. He served as a Lieutenant in the Mexican Border Dispute, and as Captain in the Infantry in France during the years 1917 to 1919.

After his separation from the United States Army, he enrolled in the University of Southern California in 1925. He became an instructor in oral surgery at the University of Southern California and later a member of the Board of Trustees of the University. Dr. Olson has practiced in Hollywood during his entire professional career.

Dr. Olson is internationally famous in the field of oral surgery and is especially well known for many technical innovations, as well as for his outstanding work in pioneering the use of thiopental sodium anesthesia in oral surgery.

Dr. Olson is a Fellow of the American College of Dentists and the Hollywood Academy of Medicine. He was one of the first oral surgeons certified by the American Board of Oral Surgeons. He is past-president of the Southern California Society of Oral Surgeons and the Hollywood Dental Society, and holds staff positions in Oral Surgery at the Children's Hospital and the Hollywood Presbyterian Hospital, Hollywood, California.

VOLUME 8 • JANUARY 1950 • NUMBER 1 JOURNAL OF ORAL SURGERY

Postextraction healing in vitamin A deficient hamsters

■ *Robert Graham Hirschi, D.D.S., Oklahoma City*

The purpose of this paper is to report the results of a study concerning the healing of postextraction wounds in vitamin A deficient animals.

Early investigators[1,2,3] reported the necessity of vitamin A for maintenance of epithelial tissues.

Hertz[4] found that fracture healing of vitamin A deficient animals was almost identical to healing on a normal diet. The biologic processes of fracture healing were almost the same although the mutual proportion in the intensity of these processes was somewhat different. In contrast Copp and Greenberg[5] have recently reported a significant delay in fracture healing in vitamin A deficient rats.

Claflin[6] found that the healing of undisturbed extraction wounds in normal dogs occurred in the following manner: hemorrhage, coagulation, thrombosis of the vessels of the alveolus, beginning clot organization, epithelial proliferation, resorption of damaged tissue and formation of new bone. He found new bone formation by the fifth day and alveolar obliteration by the oral epithelium by the seventh day. The new bone reached the crest of the ridge by the nineteenth postoperative day and healing was considered complete by the thirty-first day.

Mellanby[7] reports that vitamin A influences growth by limiting the number, and degree of activity, of osteoblasts and osteoclasts, whereas Wolbach[8] concludes that vitamin A is essential for epiphyseal cartilage growth.

Burns, Orten and Smith[9] maintained rats on a mild chronic vitamin A deficient diet for a year and noted only a slight degree of histologic change in the gingival

1. Wolbach, S. B., and Howe, P. R., Tissue Changes Following Deprivation of Fat-Soluble Vitamin A. *J. Exper. Med.* 42:753 (Dec.) 1925.

2. Mellanby, M., Periodontal Disease in Dogs. *Proc. Roy. Soc. Med. (Sect. Odont.)* 23:41 (Sept.) 1930.

3. Wolbach, S. B., and Howe, P. R., The Incisor Teeth of Albino Rats and Guinea Pigs in Vitamin A Deficiency and Repair. *Am. J. Path.* 9:275 (May) 1933.

4. Hertz, J., Studies on the Healing of Fractures. X. The Healing of Fractures in Rats on a Vitamin A Deficient Diet. *Acta. Path. et Microbiol.* 28:186 (Sept.) 1936.

5. Copp, D. H., and Greenberg, D. M., Studies on Bone Fracture Healing I. Effect of Vitamins A and D. *J. Nutrition* 29:261 (April) 1945.

6. Claflin, R. S., Healing of Disturbed and Undisturbed Extraction Wounds. *J.A.D.A.* 23:945 (June) 1936.

7. Mellanby, E., Nutrition in Relation to Bone Growth and the Nervous System. *Proc. Roy. Soc. London* 132:28 (March) 1944.

8. Wolbach, S. B., Vitamin A Deficiency and Excess in Relation to Skeletal Growth. *Proc. Inst. Med. Chicago* 16:118 (April 15) 1946.

9. Burn, C. G.; Orten, A. U., and Smith, A. H., Changes in the Structure of the Developing Tooth in Rats Maintained on a Diet Deficient in Vitamin A. *Yale J. Biol. and Med.* 13:817 (July) 1941.

Fig. 1.—Left: Deficient hamster approximately 10 weeks old, 6 weeks on diet, weight 34 Gm. Right: Control hamster 10 weeks old, 6 weeks on diet, weight 74 Gm.

tissues with some evidence of increased cornification.

In a recent experiment Glickman[10] found that root remnants of normal rats were tolerated by the adjacent tissue after the surface wound was healed. However, pathologic changes around such remnants were also demonstrated. The fact that bone fragments are exfoliated or can often be removed was explained by the involuted epithelial projections which encompass these foreign bodies.

■ *Methods*

Forty weanling hamsters 20 days old were used in this study. Eight animals acted as controls and were fed a standard dog biscuit diet (Friskies, Carnation Co., Sullivan, Wis.). The other animals were fed a diet deficient in vitamin A (Vitamin A test diet, U.S.P. No. 12). All animals were kept over wire screen and water was available at all times.

The animals were operated on the forty sixth day of the experiment. Intraperitoneal injections of a barbiturate were made and the lower left first molars were removed. Modified cotton pliers and explorers were used to extract the teeth.

The animals were sacrificed at intervals up to 20 days. A complete autopsy was performed. The head was severed from the body and a midline incision was made to separate it into halves. The left mandible was disarticulated from the skull and fixed in 10 per cent neutral formalin. The blocks of tissue thus obtained were

10. Glickman, I., Pruzansky, S., and Ostrach, M.: The Healing of Extraction Wounds in the Presence of Retained Root Remnants and Bone Fragments. *Am. J. Orthodont. & Oral Surg. (Oral Surg. Sec.)* 33:263 (April) 1947.

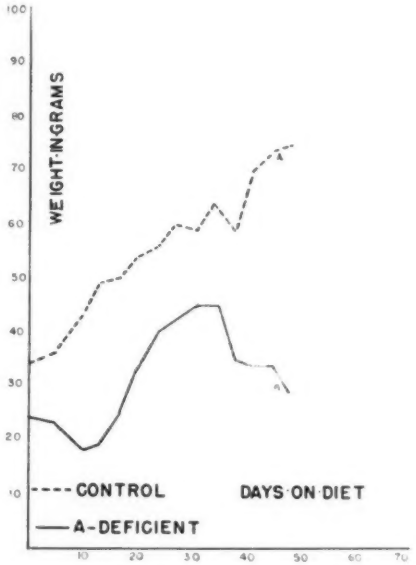


Fig. 2.—Graph showing the comparative weight curves of a deficient and control animal. The teeth were removed on the forty-sixth day (indicated by arrow) and the animals sacrificed on the forty-ninth day

decalcified in formic acid and embedded in paraffin according to the technic described by Morse.[11] Serial sections were cut in a sagittal plane at 8 microns and stained with hematoxylin and eosin.

■ *Results*

GROSS FINDINGS—The control animals remained normal and healthy throughout the experiment and continued to gain weight in every instance (Fig. 1, 2, 3, 4 and 5).

Animals deficient for 14 days showed loss of their characteristic golden color. Their hair became short, coarse and slightly thinned. Xerophthalmia affected most of them by the twenty-eighth day.

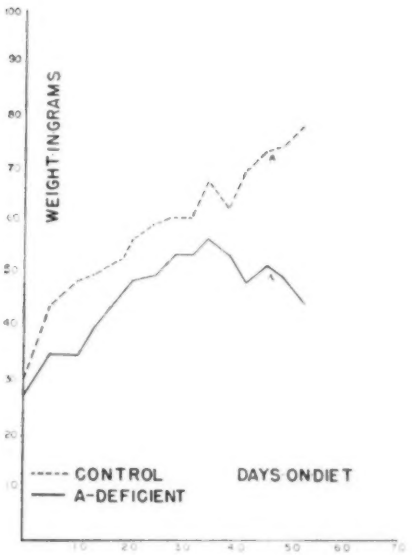


Fig. 3.—Graph showing the comparative weight curves of a deficient and control animal. The teeth were removed on the forty-sixth day (arrow) and the animals sacrificed on the fifty-third day

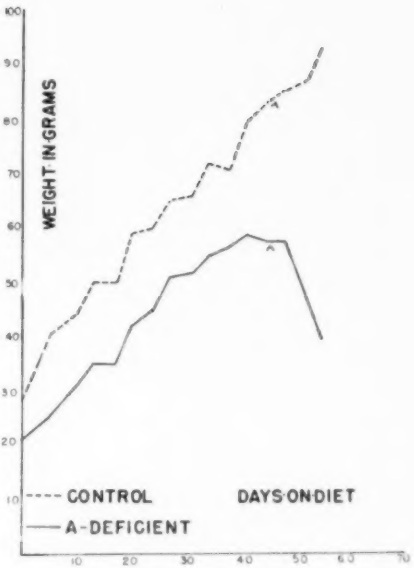


Fig. 4.—Graph showing the comparative weight curves of a deficient and control animal. The teeth were removed on the forty-sixth day (arrow) and the animals sacrificed on the fifty-sixth day

In the later stages there was hemorrhage from the genitalia and bowel and in every instance they were losing weight at the time they were killed.

MICROSCOPIC FINDINGS—Third Postoperative Day—In the control animal the oral epithelium was not intact over the crest of the alveolus. Although the top of the socket was filled with inflammatory cells, hemolyzed red cells and some bone debris, clot organization proceeded in the deeper portions. Some multinucleated

11. Morse, A.: Formic Acid-Sodium Citrate Decalcification and Butyl Alcohol Dehydration of Teeth and Bones for Sectioning in Paraffin. *J. D. Res.* 24:143 (June, Aug.) 1945.

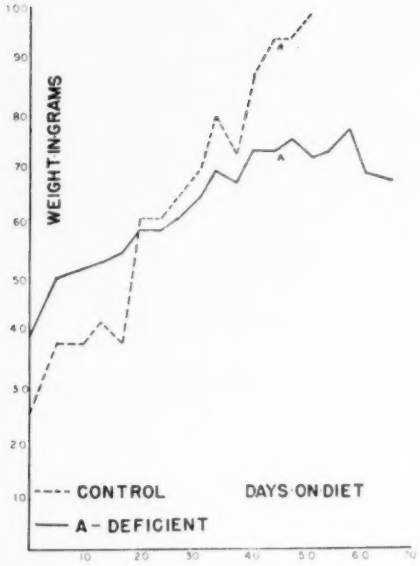


Fig. 5.—Graph showing the comparative weight curves of a deficient and control animal. The control animal weighed 114 Gm. when sacrificed. The teeth were removed on the forty-sixth day (arrow) and the animals sacrificed on the sixty-sixth day

giant cells were present (Fig. 6, *A* and *B*).

The oral epithelium in the vitamin A deficient animal did not cover the socket. There was little evidence of any alveolar outline and the operative site was filled with hemolyzed red cells, inflammatory cells and bone debris (Fig. 6, *C*).

Sixth Postoperative Day—In the control animal the oral epithelium proliferated across the top of the socket. Clot organization proceeded and many granular leukocytes were seen (Fig. 7, *A* and *B*).

In the vitamin A deficient animal the oral epithelium failed to cover the socket. There was an invagination of the oral epithelium around a mass of bone debris, clot organization was good. The alveolar bone of the adjacent molar teeth was replaced to a large degree by acute and chronic granulation tissue (Fig. 7, *C*).

Tenth Postoperative Day—New bone practically filled the alveolus in the control animal (Fig. 8, left).

In the vitamin A deficient animal the epithelium was intact. Immediately below the epithelium, osteoid tissue was present but was not as extensive as in the comparable control animal (Fig. 8, right).

Twentieth Postoperative Day—In the control animal one whole root fragment and a portion of another was retained. The pulps were necrotic in both fragments. The smaller root fragment showed areas of resorption and was lined by a few giant cells (Fig. 9, *A*).

In the vitamin A deficient animal new bone formation appeared at the top of the socket. It was not as extensive as in the comparable control (Fig. 9, *B*). The marrow and inflammatory cells, described previously, replaced practically all the alveolar bone. The oral epithelium and ameloblasts were essentially normal (Fig. 9, *C*).

■ *Discussion*

Hertz[4] has described A avitaminosis in rats. The hamsters used in our study showed similar gross changes. There was extreme weight loss and xerophthalmia was present in every animal by the time it was sacrificed. Hemorrhage from the genitalia and bowel was apparent clinically in the later stages of deficiency. In contrast to observations in other animals,[1,2,3] none of our vitamin A deficient hamsters showed evidence of change in

the oral epithelium even after 66 days on the deficient diet. The amount of epithelial cornification was considered normal (Fig. 9, *B*), and the ameloblasts of the lower incisor were essentially normal (Fig. 9, C). This latter observation differs from the conclusion of Wolbach and Howe[3] in 1933; that is, that the initial effect of vitamin A deficiency was upon the ameloblasts of the incisor teeth of rats

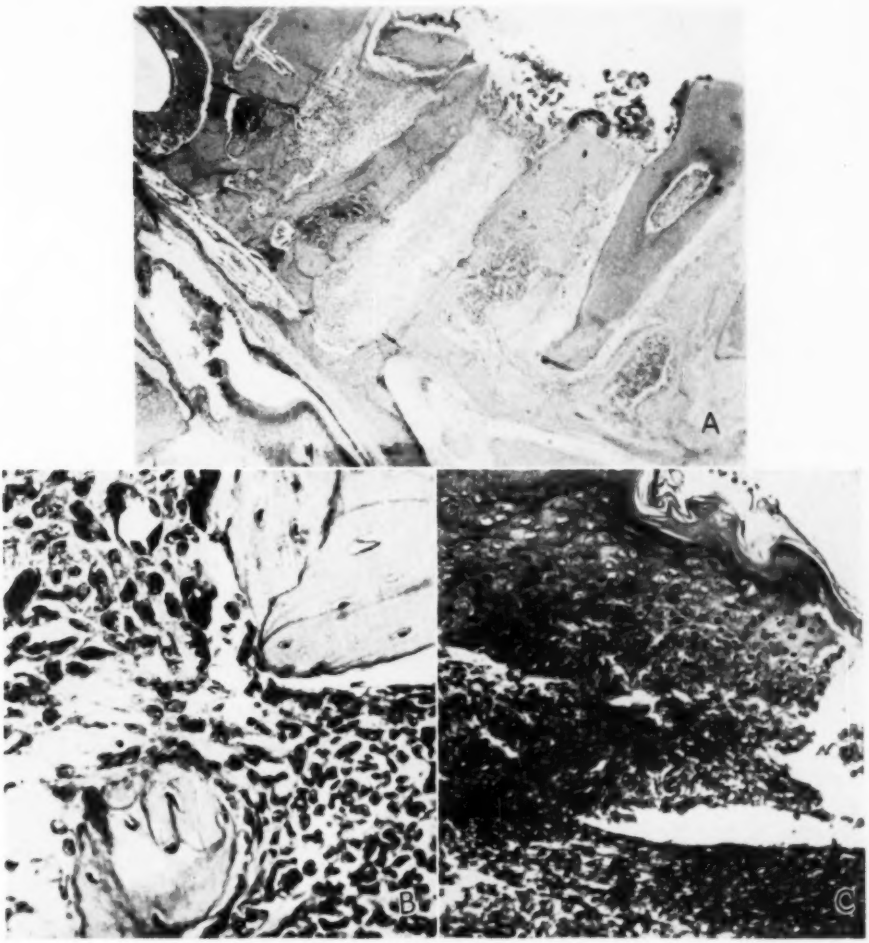


Fig. 6.—A: Three days postoperative control to show plane in which sagittal sections were cut. B: Three days postoperative control. Note the multinucleated giant cells. C: Three days postoperative deficient. Note the interrupted oral epithelium

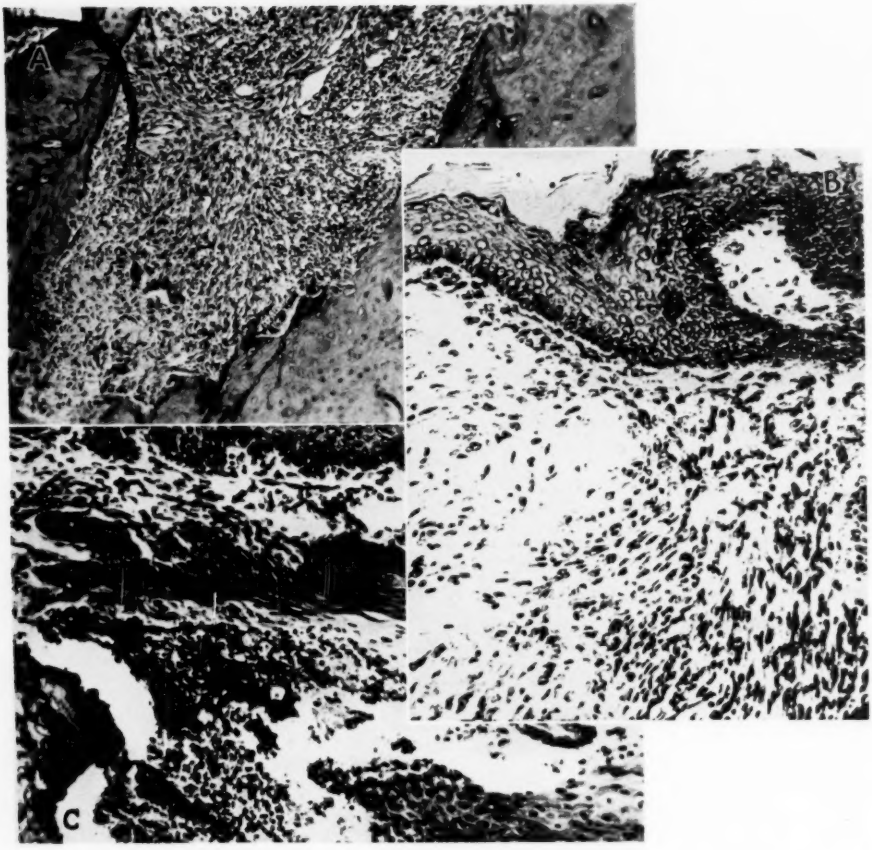


Fig. 7.—A: Six days postoperative control. Note good organization of the clot. Giant cells are seen near the bottom of the socket and along the alveolar bone. B: Notice the intact oral epithelium. Exposure made at top of socket shown in A. C: Six days postoperative deficient. Exposure made to show that the oral epithelium has not covered the socket

and guinea pigs. It would seem that the specificity of vitamin A for epithelial tissues is not particularly true for the hamster. However, further studies are indicated inasmuch as xerophthalmia was present in every instance.

Hertz[4] also noted that fracture healing in vitamin A deficient rats was almost identical with fracture healing in the animals on a normal diet. This apparently conflicts with the observations of Copp and Greenberg.[5] In the animals in their study, osteoid tissue was seen six days postoperatively in the deficient animal.

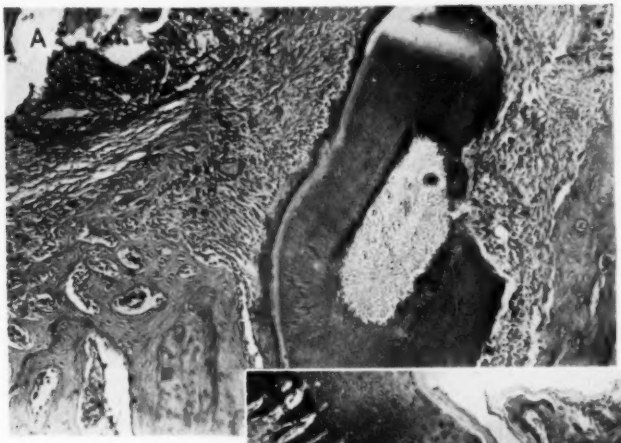


Fig. 9.–A: Twenty days postoperative control. Note resorption of retained root fragment. Osteoid tissue is seen at the left and a sequestrum of bone is in the upper left corner

B: Twenty days postoperative deficient. Note the normal, intact oral epithelium. New bone fills the top of the socket

C: Twenty days postoperative deficient. Note the normal appearance of the ameloblasts of the lower incisor

By the twentieth postoperative day new bone formation had reached the crest of the ridge in the deficient animal (Fig. 9, *B*). However, the new bone was not as extensive as in the control animal at ten days (Fig. 8, left). It would seem that the initiation of new bone is normal although quantitatively deficient.

Fibrosis of bone in both the maxillae and mandible in chronic vitamin A deficient rats with decalcification of the bony trabeculae has been reported.[9] No fibrosis was noted in the bone of the vitamin A deficient hamsters used in this study. There was evidence of extensive proliferation of bone marrow cells and acute and chronic inflammatory cells. By the twentieth postoperative day there was little differentiation between the operated socket and the adjacent alveolar bone in vitamin A deficient animals. The new bone that was present on the twentieth postoperative day appeared in very thin trabeculae. It is possible that the widespread presence of bone marrow cells and acute and chronic inflammatory cells was due to the response of the organisms to the effects of a generalized vitamin A deficiency.

Wolbach[8] has emphasized the necessity of vitamin A for epiphyseal cartilage growth. He states that appositional bone growth of periosteal origin continues until inanition supervenes at a rate of conformity with the normal growth pattern

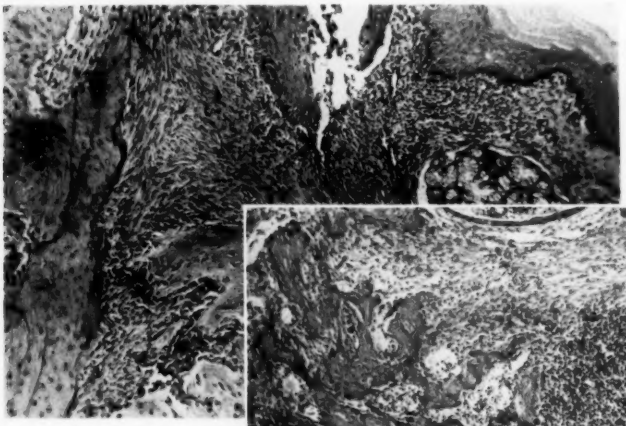


Fig. 8.—Left: Ten days postoperative control. Note the junction of the old and new bone. Photograph is at the top of the socket

Right: Ten days postoperative deficient. Note the downward proliferation of the oral epithelium. Giant cells and osteoid tissue are seen below this. In the lower right hand area of the field marrow cells and acute and chronic inflammatory cells can be seen

in each site. Our observations indicate that epiphyseal cartilage growth was arrested. Regeneration of alveolar bone did occur in the vitamin A deficient animals but not with the intensity of the controls. Apparently Mellanby[5] and Wolbach[9] agree as to the effect of vitamin A deficiency on bone growth in animals; but while Mellanby explains this effect by the influence of vitamin A on the activity of osteoblasts and osteoclasts, Wolbach believes that the effect of vitamin A deficiency is on epiphyseal cartilage and endochondral bone growth.

Claflin's[6] experiments with dogs demonstrated that new bone formation filled the crest of the socket by the twentieth postoperative day. There was new bone formation at the top of the socket in the vitamin A deficient hamster by the twentieth postoperative day (Fig. 9, *B*). The quality was normal but the quantity was insufficient.

The clinical significance of this study should be considered. The importance of clinical nutrition has been stressed repeatedly.[12] Until recently only a few animal experiments[6, 10] with possible clinical significance in dental surgery have been reported. This study of wound healing in vitamin A deficient hamsters is presented with the suggestion that the importance of good nutrition cannot be overlooked clinically. It would seem advisable to overemphasize vitamin therapy rather than neglect any essential factor.

■ *Summary and Conclusion*

A microscopic study of the healing of extraction wounds in vitamin A deficient hamsters has been presented and the literature reviewed.

Grossly the animals showed signs of vitamin A deficiency. The microscopic changes did not entirely correlate with observations reported in other vitamin A deficient animals.

The ameloblasts and oral epithelium were essentially normal. Retardation of new bone formation in the healing socket was not extreme. Initiation of new bone formation was actually seen earlier in the deficient animal than in the control. (This cannot be considered significant on the basis of one series of experiments.) The quantity of new bone, however, was decidedly less in the deficient animals than in the controls.

There was evidence of extensive proliferation of bone marrow cells and acute and chronic inflammatory cells in the alveolar bone of the deficient animals. This fact, with the accompanying decrease in quantity of old and new bone is probably the chief observation.—*514 Medical Arts Building.*

12. Bensley, E. F., The Nutrition of the Surgical Patient. *Canadian M. A. J.* 54:283 (March) 1946.

Rutin used in the oral surgery of a patient with hereditary hemorrhagic telangiectasis

■ *Charles H. Davidson, D.D.S., Bronx, N. Y.*

Hereditary hemorrhagic telangiectasis "is a vascular anomaly characterized clinically by hemorrhage and anatomically by multiple dilatations of capillaries and venules . . . in the skin and mucous membrane. Because of its clinical and familial manifestations the disorder is included among the hereditary hemorrhagic diatheses."[1]

The symptomatology consists of hemorrhage and anemia; hemorrhage occurring from the nose, tongue, mucous membrane of the mouth and gastrointestinal tract, spontaneously or due to slight trauma. "The telangiectases may be pin-point in size or large, they may form nodular vascular tumors the size of a split pea."[1]

Pathologic study shows that the walls of the blood vessels are extremely thin—sometimes containing only one layer of endothelium; or that there may be dilatation without a deficiency of elastic tissue.

Since this disease is not a blood dyscrasia but a disease of the blood vessels themselves (capillary fragility), the blood count and differential count are essentially normal except for symptoms of anemia such as a decreased erythrocyte count.

Hereditary hemorrhagic telangiectasis may be diagnosed by the triad: (1) habitual hemorrhage; (2) multiple telangiectases; (3) familial history. Until recently, the local treatment consisted of thermocautery; escharotics like silver nitrate, chromic acid and radium; snake venom; systemically O'Kane used repeated injections of 5 per cent quinine and urethane solution.[1]

The case presented here is of a patient with hereditary hemorrhagic telangiectasis whose periodontoclasia was so far advanced that it was deemed necessary to extract his remaining teeth. The danger lay in the possibility of massive blood loss due to uncontrollable hemorrhage following surgery.

Despite some reports to the effect that "operation and extraction of teeth produce no more bleeding than in normal persons,"[2] it was felt that because of the intrinsic trauma involved in multiple extractions, the possibility of massive hemorrhage was a factor to be taken into account.

■ *Report of Case*

HISTORY—A white man, aged 59, presented at the Morrisania City Hospital Dental Clinic on July 10, 1945, complaining of gingival hemorrhage and epistaxis. He could not eat well because of loose and painful teeth and profuse gingival

Clinical assistant, visiting dental surgeon, Morrisania City Hospital, Bronx, N. Y.

1. Wintrobe, M. M., *Clinical Hematology*, ed. 2. Philadelphia: Lea and Febiger, 1946.

2. Peluse, S., Gingival Hemorrhage With Oral Manifestations in Hereditary Hemorrhagic Telangiectasia; Successful Control With Sclerosing Agent Sylnasol; Effect of Vitamin P and Nicotinic Acid; Role in Case Reported Which Was Observed 5 Years. *Arch. Otolaryng.* 44:668 (Dec.) 1946.

hemorrhage. He felt that the extraction of his remaining teeth would cure his "condition," and pleaded, "Please take out my teeth."

The patient had been suffering from epistaxis as long as he could remember. Gingival hemorrhage dated back ten years. He complained of waking with a mouthful of blood. Just speaking caused hemorrhage of the gingivae. A semireclining position would bring on epistaxis. The patient had been unable to eat anything except soft foods and liquids—and even these caused pain and bleeding. He had received no treatment for his gingival hemorrhage, despite frequent hospitalizations for epistaxis and cardiac disease. His condition was diagnosed by the medical service in 1932 as hereditary hemorrhagic telangiectasis. He had lost approximately 20 to 30 pounds in the last five years.

EXAMINATION—Extraoral examination revealed a sallow-complexioned, fully developed man who seemed debilitated and undernourished. His posture was poor; he walked slowly and rested frequently. Palpation revealed no enlargement of lymph nodes. The patient was visibly depressed and hopeless and pleaded for extraction of his remaining teeth.

Intraoral examination showed: teeth present were the lower right second molar, cuspid, lateral and central incisors, left central and lateral incisors, cuspid and first bicuspid; upper central incisors and right second molar. A bridge from the lower right cuspid to the second molar was loose. All teeth showed class three mobility.

Oral hygiene was extremely poor, white matter and calculus covered all the teeth and the bridge was tarnished and crusted with white matter. The gingivae were edematous, hypertrophied, bright red in color, glossy and lacked tone. Blood clots were present around the cervical portion of all teeth and were stringy and fragile in consistency; oozing from gingival pockets was apparent.

The gingiva palatal to the upper central incisors presented two large blebs about 5 mm. in size and bright red in color. These telangiectatic blebs, when touched, hemorrhaged profusely, as did the rest of the gingivae when lightly brushed with a wisp of cotton. Telangiectatic spots appeared throughout the hard and soft palate and buccal mucosa.

Roentgenographic examination revealed extreme alveoloclasia and poorly trabeculated maxillary and mandibular bone. There were no apical radiolucencies.

The patient revealed that his mother had been a "spontaneous bleeder," and that his brother has been confined to a "chronic hospital for a long time because of his bleeding." The cause of death of the parents was not known.

Past medical history included the usual childhood diseases. The patient had been unable to work for 12 years because of a cardiac condition classified as 3C. Hemorrhage from previous tooth extractions was controlled by packing for many days.

He had been hospitalized periodically since 1931. Therapy included vitamins, tonics and numerous blood transfusions.

TREATMENT—In view of the extreme periodontoclasia and the inability of the patient to ingest food in his present condition, it was considered desirable to ex-

tract the remaining teeth so that dentures could be constructed. The patient was admitted to the hospital July 23, 1945.

A blood count at that time was normal, and the Wassermann reaction was negative. The blood picture was normal except for symptoms of secondary anemia (due to blood loss), which would indicate a disease of the blood vessels rather than of the blood itself.

The patient was placed on a high vitamin diet and given ferrous sulphate, 6 grains three times a day, calcium lactate, 10 grains three times a day, and Synkamin (vitamin K therapy), 1 ampul daily. However, after three weeks there was no improvement in the frequency of gingival hemorrhage, and multiple extractions were delayed lest massive blood loss occur.

The patient was discharged August 3, 1945, and assigned to the outpatient department, where inflammation of the gingivae was relieved temporarily by conservative curettage and the institution of mild massage with the finger. The iron, calcium and vitamin K therapy were continued, and the patient was afforded some relief. This treatment was continued until 1947.

On January 15, 1947, the patient was readmitted to the hospital for massive epistaxis, severe pains in the mouth and secondary anemia. At that time the blood count was: erythrocytes 2,600,000, leukocytes 7,400, hemoglobin 7.5 Gm. The differential white cell count was normal. There was a decrease in the platelet count. Clotting time was four minutes.

At a bedside conference between the medical and dental services on February 10, the use of rutin was discussed and the medical service advised its use in this case in order to control the hemorrhage and to enable the dental service to extract all of the remaining teeth. Rutin is a crystalline alcohol, $C_{27}H_{30}O_{16}$, from tomato stems, tobacco, rue leaves and buckwheat plants. It has the properties of vitamin P. One tablet (40 mg.) of rutin every four hours during waking hours was prescribed plus a high caloric diet.

During the next two weeks the patient improved considerably; the epistaxes became less frequent and there was no gingival hemorrhage. The large blebs palatal to the upper central incisors became firm and reddish-pink and did not hemorrhage when pressed with the finger.

On February 16 the first of the 11 extractions was performed and on March 12 the last tooth was removed. There was no postoperative hemorrhage and all the sockets healed by primary intention. Rutin was administered until the patient was discharged on March 22, 1947.

It may be of significance to state that the patient was readmitted to the hospital on July 16, with symptoms of secondary anemia. Rutin therapy was again instituted, and the patient was discharged in two weeks. This would seem to indicate that the disease can be controlled only by constant and continual use of the drug.

■ *Discussion*

Hereditary hemorrhagic telangiectasis is not a disease of the blood itself but a disease characterized by capillary fragility. Until the discovery of rutin treatment had been largely local and symptomatic—the use of rutin seems to indicate a step toward specific systemic therapy. In the case presented the primary dental problems were the oral surgery to be performed and the prevention of serious blood

loss that might endanger the patient's chances for recovery.

Rutin is derived from buckwheat leaves and blossoms, tobacco leaf, tomato stems and other plants. It is, chemically, a derivative of flavone. The flavone glucosides in association with vitamin C have been thought to regulate vascular permeability. "This factor, supposed to regulate capillary permeability also has been named vitamin P. Rutin shows neither acute nor chronic toxicity to laboratory animals, and on this basis its effects were tried on man. It was found that patients treated with daily doses of rutin for as many as 16 months showed no toxic effects attributable to the drug."[1]

■ *Conclusions*

While much research remains to be done on diseases of the blood vessels involving capillary fragility, the use of rutin made possible multiple extractions where heretofore there was an extreme reluctance to subject the patient to any form of oral surgery.

■ *Summary*

1. Hereditary hemorrhagic telangiectasis is a disease of capillary fragility.

2. A case is described in which the remaining teeth were to be removed. The fear of massive blood loss that might endanger the patient's chances for recovery was a factor for consideration.

3. Rutin, 40 mg. three times a day, was administered. This drug seems to be specific for capillary fragility, but further investigation is necessary.

4. After administration of rutin multiple extractions were performed without postoperative hemorrhage and with normal healing of the sockets.—*160 East 188th Street.*

1. Shanno, R. L., Rutin: New Drug for Treatment of Increased Capillary Fragility. *Am. J. M. Sc.* 211:539 (May) 1946.

■ Early Diagnosis of Cancer of Prime Importance

It can be stated without any hesitancy that mouth cancer is more serious and more important to the patient than any other condition with which it might be confused. Therefore, in any suspicious lesion of the mouth, the physician should consider cancer first, and should rule out such a possibility before waiting "to see what happens" or proceeding with treatment on the basis of a benign diagnosis. If the possibility of a malignant growth is considered at the beginning, few harmful errors in diagnosis will be made.—*Hayes Martin, "Cancer of the Head and Neck," American Cancer Society, Inc.*

Bone changes associated with hyperparathyroidism, Paget's disease and osteofibroma

■ *William E. Koch, Jr., D.D.S., St. Louis*

Bone lesions are often associated with systemic disturbances and may appear earliest in jaws. Many of these lesions do not give rise to clinical signs or symptoms in the early stage and are not referable to dental symptoms. Tumors and cysts of the jaws usually are not discovered until extensive destruction of bone and disfigurement has occurred. The early detection and recognition of such lesions is most important.

■ *Osteitis Fibrosa*

Generalized osteitis fibrosa (Recklinghausen's disease; osteitis fibrosa cystica) is a disease apparently due to hyperparathyroidism. Excessive parathyroid secretion, which is usually associated with hyperplasias and adenomas of one or both of the parathyroid glands, may result in generalized pathologic changes that affect all bones. These changes include resorption, deposition, fibrosis, giant cell tumors and cysts. Giant cell tumors may form rapidly, cause expansion of the bones and produce swellings. Lesions, appearing as cyst-like, giant cell tumors, may develop in the jaws first or be discovered earliest in the jaws. Tumorous formations are more prevalent in the mandible than in the maxilla. These cystic lesions are not limited to one bone but may be found scattered in various bones of a single individual. Osteitis fibrosa generally affects the middle aged and is seen more commonly in women than in men.

REPORT OF CASE—A 38 year old woman reported for examination in 1941 with the chief complaint of loose teeth and incomplete healing of the tissues where teeth had been removed. Clinical examination revealed mobility of the remaining mandibular teeth, especially the bicuspid teeth. Expansion of the cortical plates of the alveolar bone on the right side was evident. Sites of previously removed teeth showed incomplete healing of the tissues. Complete physical examination revealed swelling of the left hand and left leg. The patient attributed these swellings to arthritis and offered the information that she was under treatment for arthritis. Capsules and an ointment had been prescribed. General symptoms were loss of weight, lassitude and discomfort in the left hand and leg.

Roentgenograms were made of the teeth and supporting structures, both hands and legs. Figure 1, *A* and *B* show intraoral roentgenograms of the incisor regions of the maxilla and mandible. The bone trabeculae as seen in so-called normal alveolar bone are replaced by a reticulum of fine radiopaque lines. The radiolucent region at the apex of the maxillary right lateral incisor is the result of a degenerated pulp. The apparent increased

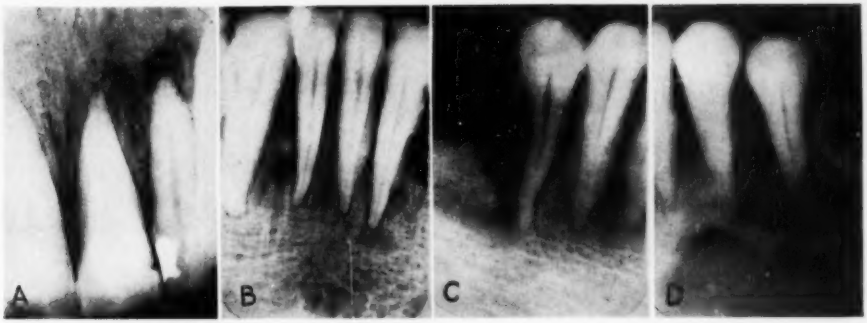


Fig. 1.—Characteristic alveolar bone changes in hyperparathyroidism. A and B: Note the reticulum of fine radiopaque lines replacing the normal bone trabeculation in the incisor areas. C and D: More striking trabeculated cyst-like areas appear in the lower bicuspid areas

density of some of the remaining mandibular teeth is probably due to demineralization of the alveolar bone.

More striking trabeculated cyst-like radiolucent regions are evident in the lower right bicuspid region and the lower left bicuspid region (Fig. 1, *C* and *D*).

Numerous cystic regions appear in the fourth metacarpal bone of the left hand, and smaller areas are present in most all phalanges (Fig. 2, *A*). The right hand (Fig. 2, *B*) presents an involvement of the second and fifth metacarpal bones.

There are multiple cystic areas involving both the medullary and cortical portions of the bone of the middle and distal third of the left tibia (Fig. 2, *C*). Similar but smaller cystic destructive

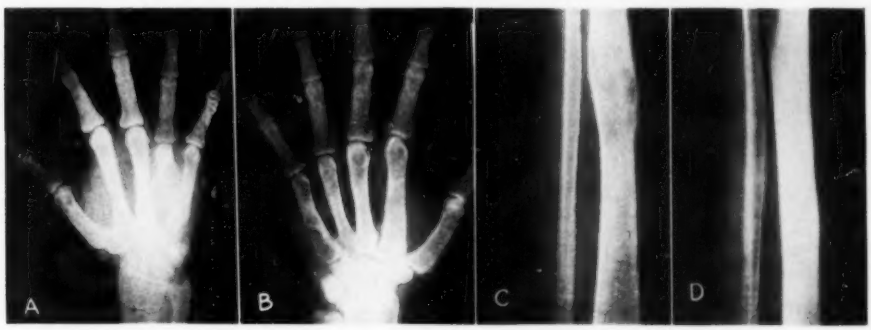


Fig. 2.—A: Numerous cystic regions appear in the fourth metacarpal bone of the left hand, and smaller areas are present in most all phalanges. B: Involvement of the second and fifth metacarpal bones in the right hand. C and D: Cystic processes involving the left and right tibia

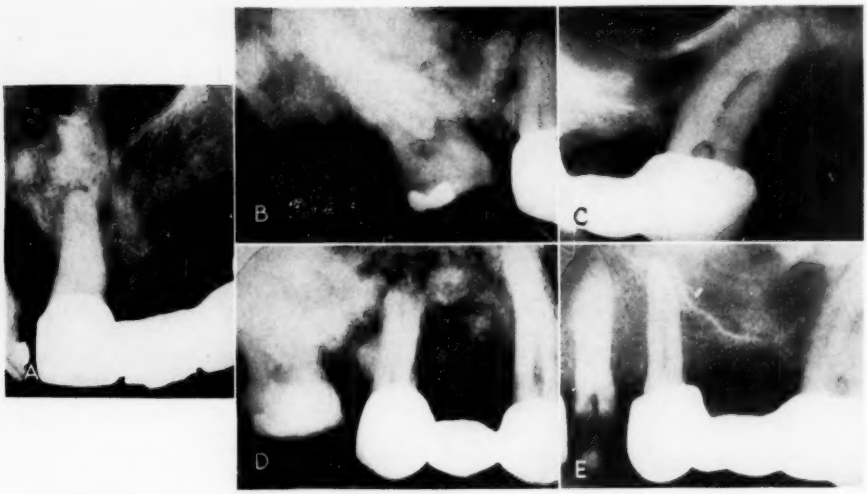


Fig. 3.—A, B, C, D: Typical "cotton wool" appearance of the alveolar bone in Paget's Disease. E: Note the apparent normal pattern of bone in the upper right bicuspid and first molar area

processes involve the proximal and middle third of the right tibia (Fig. 2, *D*).

TREATMENT—This patient was referred to an endocrinologist and a diagnosis of adenoma (benign) of the parathyroid gland was made. The adenoma was removed several months later and the patient had an uneventful recovery.

COMMENTS—All lesions of the jaws healed and the swellings of the left hand and left leg subsided. Repeated attempts have been made to secure postoperative roentgenograms, but to date all efforts have failed.

Fig. 4.—The only progressive bone change in the alveolar bone in three years

■ *Osteitis Deformans*

Paget's disease, or osteitis deformans, is a disease of the skeleton. Although a number of possible causes have been mentioned there is no definite evidence of a known etiology. Apparently this disease occurs more often in women than in men, and usually appears after middle age. It is slowly progressive but is not regarded as fatal. No special treatment has been generally accepted.

Unlike hyperparathyroidism, Paget's disease does not necessarily involve the entire skeleton. The bones involved vary according to the individual cases. Usually the maxilla is involved and rarely the mandible. According to most writers on this subject, a characteristic feature is a thickening of the calvaria. The maxilla may show changes before symptoms in the rest of the skeleton are evident. Hypercementosis may occur, especially of the bicuspid teeth. The involved surrounding alveolar bone does not seem to affect the teeth.

Apparently there is an alternating process of destruction and deposition of bone that takes place in this disease. The newly formed bone appears in excessive amounts and no doubt accounts for the thickening of the affected bones. There is a definite loss of trabecular detail in the alveolar bone. Regions of radiolucency and increased density produce the so-called "cotton wool" appearance of the bone. The maxillary arch is increased in width which tends to give the face a tapering appearance.

REPORT OF CASE—The illustrations presented are of a woman of middle age. Dental roentgenograms made in 1940 revealed the characteristic bone changes in

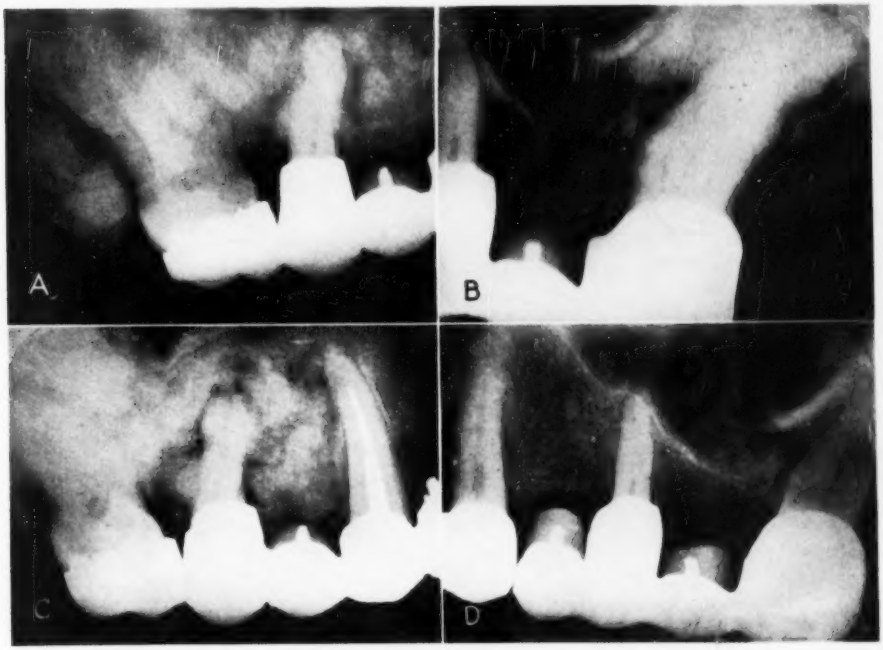


Fig. 5.—Progressive changes in all regions except upper right bicuspid and first molar region

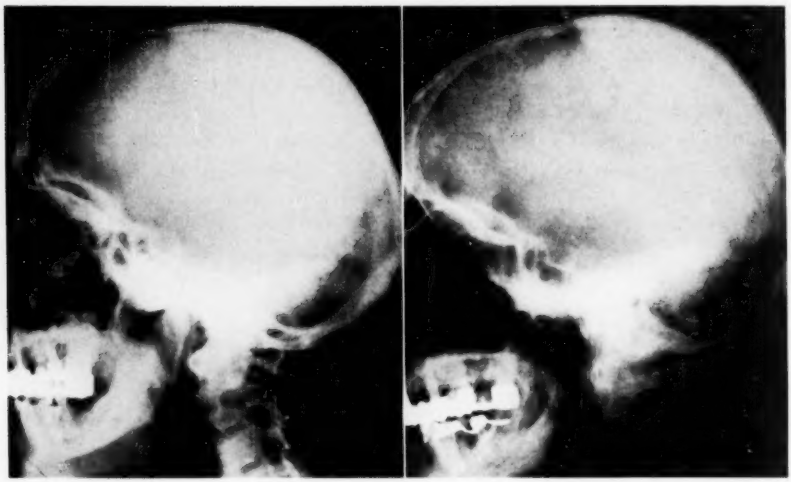


Fig. 6.—Left: No definite changes in the calvaria. Right: Some changes in the anterior portion of the calvaria

the maxillary arch. The mandible was not involved. The patient's chief complaint was pain of a neuralgic nature affecting the ear on the left side. Subsequent roentgen examinations were made in 1943, 1946 and 1948. The mandible did not show any significant changes during this period. There was a slight progressive change in the width of the maxillary arch. The neuralgic pain was absent during the last four years.

Progressive changes occurred in the alveolar bone of the maxilla. The maxillary left bicuspid and first molar regions show the typical "cotton wool" appearance. Areas of resorption and bone deposition are evident around the maxillary left second bicuspid and first molar regions (Fig. 3, *B* and *D*). Hypercementosis is noted on the distal surface of the root of the second bicuspid. The maxillary right bicuspid and first molar regions show no great abnormality (Fig. 3, *E*). There are definite areas of bone deposition above the left second molar. The incisor region shows osseous changes around the central incisor (Fig. 3, *A*). The roentgenograms depicting these changes were made in 1940.

Figure 4 illustrates progressive changes in the maxillary right second molar region, and was the only significant change evident from an examination made in 1943.

Roentgenograms made in 1946 showed progressive changes in the maxillary left bicuspid and first molar regions and the maxillary right molar region (Fig. 5). A roentgenogram of the skull revealed no significant bone disturbance (Fig. 6, left).

In 1948, intraoral roentgenograms

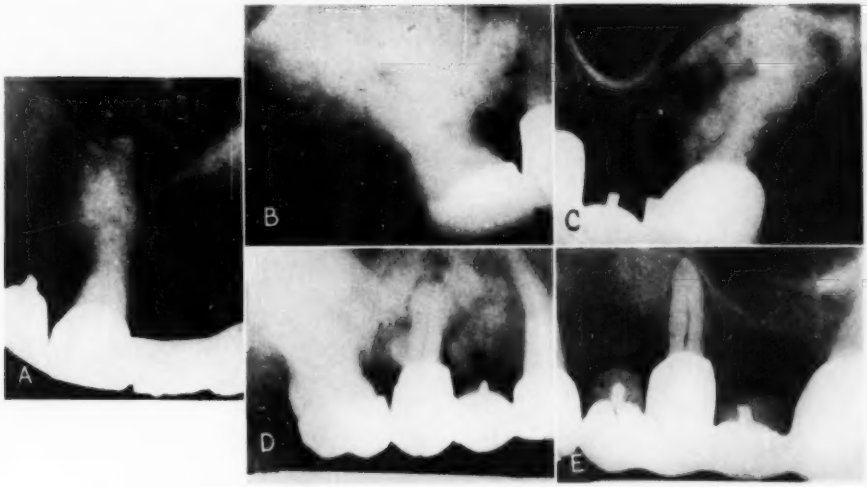


Fig. 7.—Alternating process of bone destruction and bone deposition, with distinct changes around the upper incisor. E: No changes in the upper right bicuspid and first molar area

revealed slight progressive changes in the maxillary left bicuspid, first molar and maxillary right second molar regions (Fig. 7, *B*, *C*, *D*). More noticeable changes occurred in the maxillary incisor region (Fig. 7, *A*). No changes occurred in the right bicuspid region (Fig. 7, *E*). A roentgenogram of the skull (Fig. 6, right) revealed some thickening of the calvaria, and an apparent process of bone resorption in the anterior portion of the calvaria. There was no indication of the so-called "cotton wool" or "tufted cotton" appearance in the skull.

COMMENT—There is no known treatment for osteitis deformans.

■ *Osteofibroma*

Osteofibroma (Fibro-osteoma, localized osteitis fibrosa, fibrous osteoma, fibro-osteoid-osteoma) is a benign tumor of

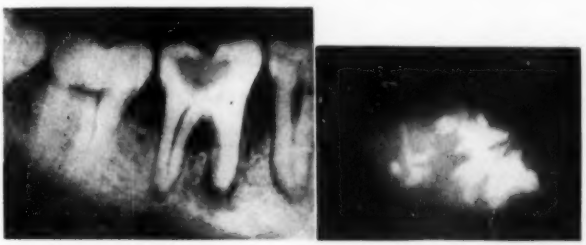


Fig. 8.—Left: Destruction of alveolar bone involving the bifurcation of the lower left first molar. Right: Roentgenogram of mass removed

osteogenic origin. The fact that this type of tumor varies greatly in the proportion of soft and hard tissues during the development and progression of the tumor mass, no doubt accounts for the various descriptive terms used. Thoma[1] prefers the term fibro-osteoma, with central ossifying fibroma, fibro-osteoma, and fibro-osteoid-osteoma to designate the various types and stages of this lesion.

This tumor produces a considerable change in the osseous structures. The maxilla or the mandible are the usual sites of involvement. The maxilla is more often involved than the mandible. The growth of this tumor is slow, and as a

1. Thoma, Kurt H.: *Oral Pathology*, ed. 2. St. Louis: C. V. Mosby Company, 1944.

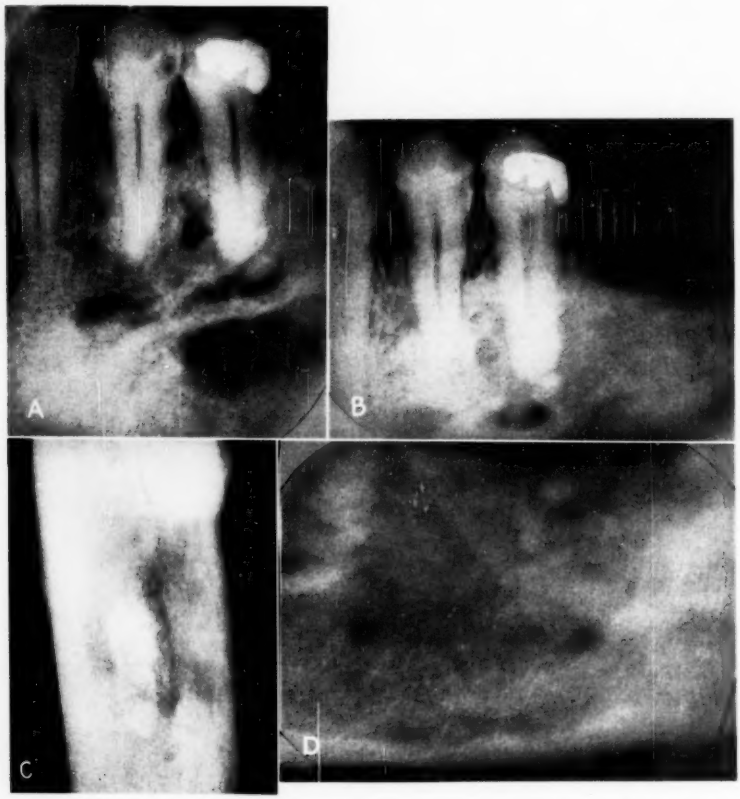


Fig. 9.—A and B: Cyst-like region around the bicuspid teeth. C: Occlusal view shows mass confined between the cortical plates. D: Normal bone trabeculation was absent in the molar region

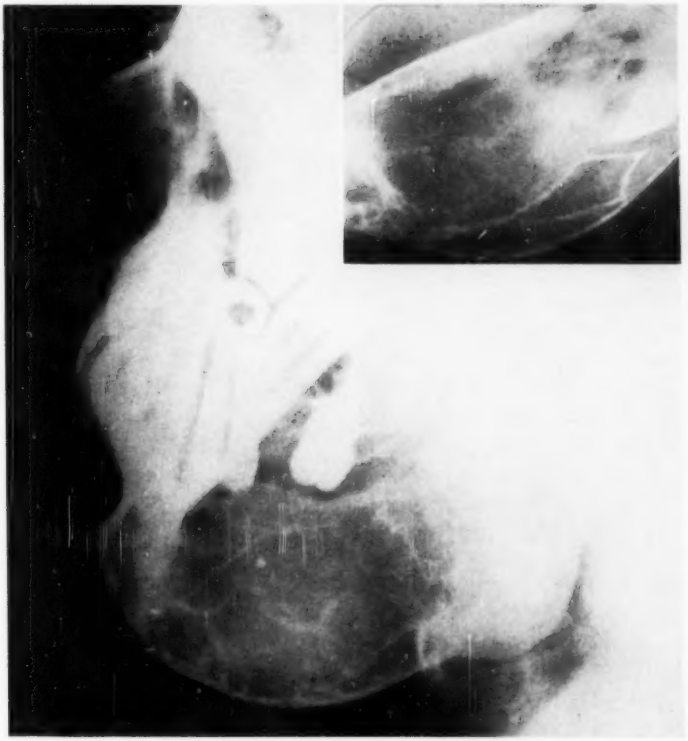


Fig. 10.—Loss of normal bone trabeculation, downward curvature of the mandible and increased width of the mandible. Occlusal view (inset) shows increased width of the mandible

rule does not produce pain. The first symptoms generally are disfigurement of the region involved and disturbance of the occlusion.

CASE 1—Patient presented with chief complaint of "abscessed tooth." Clinical examination revealed inflammation of the soft tissues around the lower left first molar. Extension of the lingual cortical plate was noted. The first molar was tender on percussion and did not respond to vitality tests.

Roentgenographic examination revealed chronic periapical bone involvement, especially noticeable around the mesial portion of the root. Destruction of marginal bone support around the tooth and in the bifurcation of the roots was evident (Fig. 8, left). The tooth was removed, as well as the semihard mass on the lingual surface. Figure 8, right, shows

a roentgenogram of this mass. Biopsy revealed a formation of bone and fibrous tissue, and the diagnosis of osteofibroma was made.

CASE 2—A woman patient complained of discomfort in the lower jaw. Clinical examination revealed no signs of swelling or extension of the cortical plates of bone. A well constructed partial denture was excluded as a possible source of irritation. The cuspid and bicuspid teeth responded normally to vitality tests. The soft tissues appeared normal.

Roentgenograms revealed a distinct change in the structure of the alveolar bone. Small cyst-like regions were noted around and between the bicuspid teeth (Fig. 9, *A* and *B*). There was no sharp line of demarcation between the involved area and the surrounding alveolar bone. The occlusal view showed the mass confined between the cortical plates of the bone (Fig. 9, *C*). The normal bone trabeculation was absent in the molar region (Fig. 9, *D*). A biopsy was performed and a diagnosis of osteofibroma was reported.

CASE 3—Clinical examination of a boy, aged 14, revealed a considerable extension of the cortical plates of the mandible and the soft tissue over the alveolar ridge. The maxillary teeth occluded into the soft tissues when the jaws were closed. No pain was present. The patient gave a history of swelling in this region over a long period of time, with the gradual loss of the teeth involved. No roentgenograms were made until disfigurement was noted. The roentgenograms revealed a loss of normal trabeculation, distinct increase in the width of the mandible and a downward curvature of the inferior border of the mandible (Fig. 10). The mass was semihard and no pain was evident upon pressure. The diagnosis, based upon biopsy, was fibro-osteoid-osteoma.

■ *Summary*

These cases are presented mainly to stress the importance and value of adequate routine roentgenographic examinations. Often the earliest symptom of bone disease may be manifest in the jaws. Many of these bone lesions could be discovered earlier if roentgenographic examinations were more generally used. The absence of clinical signs or symptoms does not mean that roentgenograms are unnecessary, because bone lesions and diseases of the jaws are not necessarily referable to dental symptoms. The earlier the recognition, the more favorable is the prognosis.

It is not the intent of this paper to infer that the cases presented can be diagnosed from roentgenograms alone, but that the possibility of such lesions should be suspected and further investigations be recommended.—*615 Metropolitan Building.*

Gingival hyperplasia resulting from diphenylhydantoin sodium: a review of the literature

■ *Zar A. Reader, D.D.S., Detroit*

Diphenylhydantoin sodium ("Dilantin") is the drug which has proved most beneficial in the control of epileptic seizures. In using this therapeutic agent, certain side actions may occur, the most prominent being a gingival hyperplasia.

Christian[1] defines epilepsy as "an affection of the nervous system, characterized by attacks of unconsciousness, with or without convulsions. It is more a clinical syndrome than a disease. The transient loss of consciousness without convulsive seizures is known as minor epilepsy, 'petit mal'; the loss of consciousness with general convulsive seizures is known as major epilepsy, 'grand mal.' Localized convulsions, occurring usually without loss of consciousness, are known as 'Jacksonian' or 'cortical epilepsy'."

Epilepsy, an ancient disorder, was formerly looked upon with despair by the medical profession. There were many remedies but they were discouragingly unbeneficial. In 1857 bromides were used for the first time by Locock.[2] Phenobarbital, introduced under the proprietary name "Luminal," was first advocated by Hauptmann[3] in 1912, and displaced the bromides as the chief drug used in epileptic treatment. When phenobarbital was prescribed there was often present a dull, drowsy, stupefying feeling, while the bromides caused rashes and mental dullness.[4] Next, research was concerned with the ketogenic diet[5] and the restriction of fluids[6] which gave few beneficial results. Lesions of the brain which cause epileptic attacks can be removed by surgical procedures; also surgical excision of cortical scars or irritable cortical foci can be done.[7]

However, with all of these methods of treatment, a great number of patients are not relieved of their attacks, or, if they are aided in elimination or reduction in the number of their seizures, often the benefit is only temporary.[8]

■ *History of Diphenylhydantoin Sodium*

Merritt and Putnam[9] began a new era in the treatment of epilepsy with their introduction of the drug diphenylhydan-

1. Christian, H. A., *Principles and Practice of Medicine*, ed. 16. New York: Appleton-Century-Crofts Co., Inc., 1947.
2. Locock, C., Discussion of Paper, Analysis of 52 Cases of Epilepsy Observed by the Author, by E. H. Sieveking. *Lancet* 1:528, 1857.
3. Hauptmann, A., Luminal bei Epilepsie. *Munchen med. Wchnschr.* 59:1907, 1912.
4. Merritt, H. H., Recent Advances in Treatment of Epilepsy. *New York State J. Med.* 46:2629 (Dec.) 1946.
5. Geyelin, H. R., Fasting as a Method for Treating Epilepsy. *M. Rec.* 99:1037 (June) 1921.
6. Fay, T., The Therapeutic Effect of Dehydration on Epileptic Patients. *Arch. Neurol. & Psychiat.* 23:920 (May) 1930.
7. Merritt, H. H., The Treatment of Epilepsy. *J. Mt Sinai Hosp.* 14:849 (Nov.-Dec.) 1947.
8. Merritt, H. H., and Putnam, T. J., Sodium Diphenyl Hydantoinate in the Treatment of Convulsive Disorders. *J.A.M.A.* 111:1068 (Sept.) 1938.
9. Merritt, H. H.; Putnam, T. J., and Schwab, D. M., A New Series of Anti-Convulsant Drugs Tested by Experiments on Animals. *Arch. Neurol. & Psychiat.* 39:1003 (May) 1938.

toin sodium. More than 700 synthetically produced compounds which the investigators thought might possibly have an anticonvulsant effect were studied. Diphenylhydantoin sodium was the drug of choice and, in May 1938, it was shown to have a highly anticonvulsant effect against electrically induced convulsions in laboratory animals. In September 1938, good results against clinical epilepsy were obtained by Merritt and Putnam.[8]

Diphenylhydantoin sodium is commonly called "Dilantin," but many other names are used, such as "Dilantin Sodium"; sodium diphenylhydantoinate; phenytoin sodium; sodium 5, 5-diphenylhydantoinate; 5, 5-diphenyl glycolurea, and "Epanutin" (Great Britain).

The formula for diphenylhydantoin sodium is $C_{15}H_{11}N_2O_2Na$.[8]

"Dilantin," the trade name of the drug produced by Parke, Davis & Company, is a derivative of glycol urea and structurally is analogous to the barbiturates. It is an odorless, white powder with a bitter taste, is soluble in water, and forms an alkaline solution with a pH of 11.7.[10]

Capsules containing the powder are supplied, and also "Dilantin in Oil" for patients who have gastric irritation after the conventional method of administration. The dosage[8, 10] is one (1½ grains or 1/10 Gm.) to six capsules per day as needs dictate, that is, enough to control seizures adequately. The correct dosage is different for every individual and is determined by trial. If more than six capsules are given, toxic manifestations usually occur.

The drug usually is contraindicated in aged patients with hypertension, cardiorenal disease, or in debility.[11] However, it is considered safe, for it is almost impossible for a patient to take a lethal dose.[7] Only two fatalities have been reported[12, 13] as due directly to the toxicity of the drug.

Clinical tests by investigators quickly showed that diphenylhydantoin sodium was the most powerful anticonvulsant. Some of the early results are shown in Table 1. All age groups were included, except in the studies by Kimball, Hodgson and Reese, which were for children.

To gain even higher degress of suc-

10. Robinson, L. J., Evaluation of Continued Therapy with Phenytoin Sodium. *Am. J. Psychiat.* **99**:231 (Sept.) 1942.

11. Morgan, J. A., Dilantin (Sodium Diphenylhydantoinate) in Treatment of Epilepsy. *Illinois M. J.* **77**:542 (May) 1940.

12. Kimball, O. P., and Horan, T. N., Use of Dilantin in Treatment of Epilepsy. *Ann. Int. Med.* **13**:787 (Nov.) 1939.

13. Ritchie, E. B., and Kolb, W., Reaction to Sodium Diphenyl Hydantoinate (Dilantin Sodium). Hemorrhagic Erythema Multiforme Terminating Fatally. *Arch. Dermat. & Syph.* **46**:856 (Dec.) 1942.

Table 1
Results of investigations for control of grand mal with diphenylhydantoin sodium

Year	Investigator	Number of patients	Percentage of seizures completely or greatly relieved
1938	Merritt & Putnam[8]	142	85
1939	Kimball[14]	129	77
1939	Kimball & Horan[12]	220	75
1939	Hodgson & Reese[15]	88	66
1940	Allen[16]	65	69
1940	Frankel[17]	48	60
1940	Ross & Jackson[18]	73	65
1942	Robinson[10]	41	63

cess in the control of seizures, diphenylhydantoin sodium in combination with phenobarbital may be tried.[19]

The control of petit mal seizures with diphenylhydantoin sodium has proved to be excellent[16] but not as uniformly successful as with grand mal. Many researchers write that the beneficial effect of the drug is greater for children than for adults who have been chronic epileptics.[20, 21] Merritt and Putnam[22] demonstrated it to be 79 per cent more effective than all other methods of treatment.

Because the number of convulsive attacks is lessened and because this drug has practically no hypnotic effect in therapeutic doses, the patients gain in many ways. There is great improvement in general health, mental attitude, personality, behavior, higher learning and gain in weight. Patients are able to resume partially their places in society according to their physical and mental ability.[10, 8, 14, 18]

In 1946 Merritt and Brenner[23] stated, "The efficacy of sodium 5, 5-diphenylhydantoinate in preventing convulsive seizures in humans has been confirmed by many observers, and it is the most effective agent for the prevention of grand mal and psychomotor seizures that is available at the present time." It is regarded as a salvation to the epileptic because such great numbers of epileptic patients are now being helped that were never helped before.

■ *Side Actions of Diphenylhydantoin Sodium Therapy*

In controlling epileptic seizures with diphenylhydantoin sodium, often untoward actions occur which are sometimes annoying, but rarely contraindicate the continued use of the therapeutic agent. If the optimum dosage is prescribed, the side actions seldom occur, but when they do occur they are usually mild.

Most investigators report a gingival hyperplasia or hypertrophy. The nervous system is often attacked and tremors, vertigo, ataxia and a feeling of uneasiness may result.[24] When insomnia and irritability result, phenobarbital often is added to the dosage. Sometimes blurring of vision[8] and diplopia occur. With disturbance of the gastrointestinal system[12, 22] the patient may have anorexia, slight nausea, vomiting and loss of weight. If the gastrointestinal irritability is caused by the alkalinity of the drug, it can be given at mealtime or in oil. Merritt and Putnam[8] reported a 5 per cent dermatitis. The consensus stated little change in blood counts or blood pressure; noticeable changes were shown in electrocardiograms.[25]

14. Kimball, O. P., Treatment of Epilepsy with Sodium Diphenyl Hydantoinate. *J.A.M.A.* 112:1244 (Apr.) 1939.

15. Hodgson, E. R., and Reese, H. H., Clinical Experience with Dilantin in Epilepsies. *Wisconsin M. J.* 38:968 (Nov.) 1939.

16. Allen, F. N., Improved Treatment of Epilepsy with Dilantin Sodium. *Lahey Clin. Bull.* 1:20 (Apr.) 1940.

17. Frankel, S. I., Sodium Dilantin in Treatment of Epilepsy. *J.A.M.A.* 114:1320 (Apr.) 1940.

18. Ross, A. T., and Jackson, V., Dilantin Sodium; Its Influence on Conduct and on Psychometric Ratings of Institutionalized Epileptics. *Ann. Int. Med.* 14:770 (May) 1940.

19. Robinson, L. J., and Osgood, R., Comparative Effects of Phenobarbital and Dilantin Sodium in Treatment of Epilepsy. *J.A.M.A.* 114:1334 (Apr.) 1940.

20. Blair, D.; Bailey, K. C., and McGregor, J. S., Treatment of Epilepsy with Epanutin. *Lancet* 2:363 (Aug.) 1939.

21. Steel, J. P., and Smith, E. S., Epanutin in Epilepsy. *Lancet* 2:367 (Aug.) 1939.

22. Merritt, H. H., and Putnam, T. J., Sodium Diphenyl Hydantoinate in Treatment of Convulsive Seizures; Toxic Symptoms and Their Prevention. *Arch. Neurol. & Psychiat.* 42:1053 (Dec.) 1939.

23. Merritt, H. H., and Brenner, C., Studies in New Anticonvulsants. *Proc. A. Research Nerv. & Ment. Dis.*, 1946.

24. Fetterman, J. L., Dilantin Sodium Therapy in Epilepsy. Report of Study in Progress. *J.A.M.A.* 114:396 (Feb.) 1940.

25. Finkelman, I., and Arieff, A. J., Untoward Effects of Phenytoin Sodium in Epilepsy. *J.A.M.A.* 118:1209 (Apr.) 1942.

None of these secondary reactions should constitute a danger to the patient, provided he is under adequate medical and dental supervision.[10]

If side actions develop, they frequently disappear with a reduction or brief suspension in dosage, and sometimes they do not recur with a restoration of the dosage that will adequately control seizures. If a reduced dosage must be used, a combination with phenobarbital often will control seizures.[10, 26]

In Merritt and Putnam's article in 1938,[8] 200 patients were studied, but no mention was made of gingival change. However, Kimball's report[14] on 119 patients soon followed, in which he noted that 57 per cent of his patients had gingival hyperplasia. Most of the succeeding reports recognized gingival hyperplasia or, as many stated, "gingival hypertrophy."[27]

Table 2 shows the high incidence of gingival hyperplasia as a result of the use

26. Stern, L.; Eisenbud, L., and Klatell, J., Analysis of Oral Reactions to Dilantin Sodium. *J. D. Res.* **22**:157 (Apr.) 1943.

27. Robinson, L. J., Gingival Changes Produced by Dilantin. *Dis. Nerv. System* **3**:88 (Mar.) 1942.

Table 2

Incidence of gingival hyperplasia occurring in patients receiving diphenylhydantoin sodium

Year	Investigator	Number of Patients	Percentage showing gingival changes
1938	Merritt, Putnam[8]	142	Not mentioned
1939	Philips[28]	12	Not mentioned
1939	Kimball[14]	119	57
1939	Kimball, Horan[12]	220	51
1939	Blair, Bailey, McGregor[29]	75	2.5
1939	Steel, Smith[21]	20	15
1939	Frost[29]	9	Not mentioned
1939	Weaver, Harrell, Arnold[30]	14	Not mentioned
1939	Pratt[31]	52	50
1939	Williams[32]	91	Not mentioned
1939	McCarton, Carson[33]	20	5
1939	Hodgson, Reese[15]	44	7
1939	Merritt, Putnam[22]	350	6
1940	Fetterman[23]	28	25
1940	Weinberg, Goldstein[34]	15	33
1940	Robinson, Osgood[19]	100	Percent not given
1940	Allen[16]	65	Not mentioned
1940	Frankel[17]	48	62.5
1940	Ross, Jackson[18]	73	Percent not given
1940	Morgan[11]	8	Percent not given
1940	Johnson[35]	20	Not mentioned
1940	Merritt, Foster[36]	182	22
1940	Butter[37]	43	2
1940	Schlotthauer[24]	32	13
1941	Lowry[39]	34	29
1941	Glickman, Lewitus[40]	76	21
1942	Millhon, Osterberg[41]	30	40
1942	Robinson[27]	143	19
1942	Macfarlane, Baxter, Mitchell[42]	67	55
1942	Prudhomme[43]	57	30
1942	Polk[44]	20	50
1943	McLendon[45]	29	3.5
1943	Stern, Eisenbud, Klatell[26]	50	52
1945	Esterberg, White[46]	244	54
1946	Bashinski[47]	30	Percent not given

of diphenylhydantoin sodium in the treatment of epilepsy. It would seem that there is a discrepancy in the percentages which are extremely low.[28] Also, several investigators made no mention of their detection of gingival change, but they must have been aware of the possibility of gingival abnormality. Stern, Eisenbud and Klatell[26] suggested that this disparity possibly was caused by frequent, subtle gingival changes which were overlooked, particularly by the clinician who was not thoroughly familiar with the appearance of normal periodontal tissues.

The terms "hyperplasia" or "hypertrophy", or both,[42] have been used by the investigators to describe the gingival overgrowth resulting from the use of diphenylhydantoin sodium. In the *Classification of Periodontal Diseases* in 1942,[48] which was recommended by the Nomenclature Committees of the American Dental Association[49] and of the American Academy of Periodontology,[50] hypertrophy was the principal term used to designate overgrowth of the gingival tissue which was characteristic of certain cases, including the drug action of diphenylhydantoin sodium.

In 1944, Miller[51] wrote that hypertrophy is an increase in the size of the individual cells, as a result of which, an organ may become enlarged. Hyperplasia is an increase in size resulting from an increase in the number of cells of an organ. Hyperplasia, unlike hypertrophy, is often the result of irritation. According to these definitions probably either or both hyperplasia and hypertrophy occurred in the diphenylhydantoin sodium overgrowth.

The Report of the Committee on Nomenclature of the American Academy of Periodontology[52] in 1947 altered these definitions. Orban[53] described hypertrophy as an overgrowth of an organ by enlargement of its specific tissue elements, an enlargement which serves a useful function. Hyperplasia refers to an enlargement of an organ caused by multiplication of its structural elements or to an accumu-

28. Philips, D. P., Epilepsy as Prison Problem: Its Treatment with Sodium Diphenylhydantoinate. *J. Michigan M. Soc.* **38**:317 (Apr.) 1939.
29. Frost, I., Sodium Diphenyl Hydantoinate in Treatment of Epilepsy. *J. Ment. Sc.* **85**:976 (Sept.) 1939.
30. Weaver, O. M.; Harrell, D. L., and Arnold, G. B., Sodium Diphenyl Hydantoinate in 15 Selected Cases of Epilepsy. *Virginia M. Monthly* **66**:522 (Sept.) 1939.
31. Pratt, C. H., Sodium Diphenyl Hydantoinate (Dilantin) and Its Combination with Phenobarbital in the Treatment of Epilepsy. Review and Preliminary Report. *J. Ment. Sc.* **85**:986 (Sept.) 1939.
32. Williams, D., Treatment of Epilepsy with Sodium Diphenyl Hydantoinate. *Lancet* **2**:678 (Sept.) 1939.
33. McCarton, W., and Carson, J., Use of Sodium Diphenyl Hydantoinate. *J. Ment. Sc.* **85**:965 (Sept.) 1939.
34. Weinberg, J., and Goldstein, H. H., Comparative Study of Effectiveness of Dilantin Sodium and Phenobarbital in Group of Epileptics. *Am. J. Psychiat.* **96**:1029 (Mar.) 1940.
35. Johnson, H. K., Responses of Various Types of Epilepsy to Dilantin Therapy. *Psychiatric Quart.* **14**:612 (July) 1940.
36. Merritt, H. H., and Foster, A. M., Vitamin C in Epilepsy. Dilantin Sodium Not a Cause of Vitamin C Deficiency. *Am. J. M. Sc.* **200**:541 (Oct.) 1940.
37. Butter, A. J. M., Epilepsy Treated with Epanutin. *Brit. M. J.* **1**:483 (Mar.) 1940.
38. Schlotthauer, C. E., Dilantin Sodium Therapy of the Convulsive Disorders: 2 Year Study. *Washington Bull.* **3**:16 (July) 1940.
39. Lowry, E. C., Dilantin Therapy in Epileptiform Seizures in Children. *Lancet* **61**:178 (May) 1941.
40. Glickman, I., and Lewitus, M. P., Hyperplasia of Gingivae Associated with Dilantin (Sodium Diphenyl Hydantoinate) Therapy. *J.A.D.A.* **28**:199 (Feb.) 1941.
41. Millhon, J. A., and Osterberg, A. E., Relationship Between Gingival Hyperplasia and Ascorbic Acid in Blood and Urine of Epileptic Patients Undergoing Treatment with Sodium 5, 5-Diphenyl Hydantoinate. *J.A.D.A.* **29**:207 (Feb.) 1942.
42. Macfarlane, T. W.; Baxter, H., and Mitchell, D. A., Some Observations on Epanutin Therapy in Epilepsy. *Glasgow M. J.* **138**:189 (Dec.) 1942.
43. Prudhomme, C., Dilantin Sodium in Epileptic Negroes. *J. Nat. M. A.* **34**:150 (July) 1942.
44. Polk, D. S., Treatment of the Epileptic Child. *Pennsylvania M. J.* **45**:1293 (Sept.) 1942.
45. McLendon, S. B., Comparative Study of Dilantin Sodium and Phenobarbital in Negro Epileptics. *South. M. J.* **36**:303 (Apr.) 1943.
46. Esterberg, H. L., and White, P. H., Sodium Dilantin Gingival Hyperplasia. *J.A.D.A.* **32**:16 (Jan.) 1945.
47. Bashinski, B., Treatment of Epilepsy in Children with Sodium Dilantin. *J. M. A. Georgia.* **35**:228 (Aug.) 1946.
48. Orban, B., Classification and Nomenclature of Periodontal Diseases. *J. Periodont.* **13**:88 (July) 1942.
49. Report of Nomenclature Committee. *J.A.D.A.* **30**:1112 (July) 1943.
50. Report of Nomenclature Committee of the American Dental Association. *J. Periodont.* **14**:14 (Oct.) 1943.
51. Miller, S. C., *Textbook of Periodontia*, ed. 2. Philadelphia: Blakiston Company, 1944.
52. Nomenclature in Periodontia. *J. Periodont.* **18**:174 (Oct.) 1947.
53. Orban, B., Periodontal Disturbances; Practical Management. *J.A.D.A.* **35**:543 (Oct.) 1947.

lation of foreign elements, such as fluid and inflammatory cells, an enlargement which does not necessarily serve an increased and useful function of the organ. It is obvious from these definitions that hyperplasia is the correct term for the description of a gingival overgrowth resulting from diphenylhydantoin sodium, or perhaps any gingival overgrowth.

■ *Cause of "Dilantin" Gingival Hyperplasia*

Kimball,[33] was the first to recognize gingival hyperplasia accompanying diphenylhydantoin administration. In 1939 he prescribed ascorbic acid and a high vitamin C diet to 34 epileptic children receiving the drug, but the analysis of the blood serum showed the ascorbic acid level to be low. He saw that no patient with normal gingival tissues had a low ascorbic acid blood level. The more serious the gingival hyperplasia, the higher was the ascorbic acid deficiency. Therefore, he concluded that the amount of hyperplasia was proportional to the deficiency of ascorbic acid. However, he did not see the usual symptoms of severe and prolonged vitamin C deficiency.

A few investigators[17, 38] agreed with Kimball, but soon the theory was disproved conclusively,[34, 41, 55, 56] the only parallel being that the hyperplasia resembled scurvy in a superficial way.[55] The constitutional signs of vitamin C deficiency that were absent were purpura, sore joints and extreme weakness; also missing were the periodontal manifestations of pain, marginal necrosis, petechial hemorrhage and loosening of the teeth. Others[56] observed no difference in the plasma vitamin C levels of patients who had never received diphenylhydantoin sodium and those who had been receiving it for two years. Many showed that they could produce no diet inadequacies which would yield hyperplasia.[28]

Diphenylhydantoin sodium is the only drug used in epileptic treatment that causes gingival hyperplasia.[46]

With the withdrawal of the drug, there is a gradual disappearance of the gingival abnormality over a period of several weeks or longer.[27] At a later date, it again may be prescribed and usually the hyperplasia will return. With a constant dosage there is no predictable course in the onset, progression, or regression of the hyperplasia,[27, 56] thus the gingivae must be watched carefully during administration of the drug. Stern, Eisenbud and Klatell[26] noted that the longer the period of therapy lasted, the larger was the incidence and the larger was the degree of hyperplasia. Macfarlane, Baxter and Mitchell[42] reported that 34 per cent of their patients manifested hyperplasia early in treatment; a year later, 55 per cent of the same group had gingival overgrowths.

The constitutional tendency to produce hyperplasia varies with the individual. Why one person has this systemic tendency and another person has not, cannot be satisfactorily explained.[40] Clinical evidence points to endocrine disturbances in the background of a great num-

34. Dilantin Sodium; Report of Council on Pharmacy and Chemistry. *J.A.M.A.* 113:1734 (Nov.) 1939.

55. Ziskin, D. E., Stowe, L. R., and Zegarelli, E. V., Dilantin Hyperplastic Gingivitis; Its Cause and Treatment; Differential Appraisal. *Am. J. Orthodont. & Oral Surg. (Oral Surg. Sec.)* 27:350 (July) 1941.

56. Thompson, E. C., and Gillespie, J. B., Hyperplasia of Gums Following Dilantin Therapy, with Gingivectomy for Correction. *J.A.D.A.* 28:1613 (Oct.) 1941.

ber of cases, but these cannot be clearly identified.[46] Racial extraction may play a role, for there were 25 per cent more cases of hyperplasia among the patients that were predominantly dark and had an excess of body hair than in the average group.

In determining the incidence of gingival reaction, most investigators showed that there was no correlation with age, sex, length of treatment, degree of therapeutic effectiveness of diphenylhydantoin sodium, size of optimum dosage, weight or height, but rather with the individual tolerance or sensitivity to the drug.[46, 28, 55]

Frankel[17] thought that the larger the dose, the greater the hyperplasia. This often may be true if the dosage is larger than the optimum for the patient.

Robinson[27] believed the highest incidence of overgrowth is found in young age groups; other investigators[42, 55] showed that gingivitis is common among epileptic patients, especially children, regardless of the treatment used, because of lack of oral hygiene.

There has been considerable controversy as to whether local factors, such as calculous deposits, oral sepsis, ill designed restorations (overhanging cervical margins, poor contact points), unfilled cervical cavities, irregular dentition and traumatic occlusion are the direct or indirect etiologic factors of the gingival tissue overgrowth, or whether local conditions have no relation.

Ziskin, Stowe and Zegarelli[55] suggested that, because of gingival inflammation caused by local conditions, stasis is produced in the terminal capillaries, thus making possible the action of diphenylhydantoin sodium in producing hyperplasia.

Esterberg and White,[46] with 244 patients, reported that although local conditions were such that infection was invited, no Vincent's angina was noted. Also, they noted that all patients with an open bite had hyperplasia. Macfarlane, Baxter and Mitchell[42] believed prevalence of mouth breathing aided gingivitis.

All investigators, except a few[41, 46] believed that local factors affect the degree of hyperplasia. Stern, Eisenbud and Klatell[28] reported that the classic picture of "Dilantin" hyperplasia shows an overgrowth of gingival tissue with no inflammation. When the classic picture is complicated with local irritants, the result is an overgrowth, plus a superimposed inflammation. The common local irritants which impair local resistance act as aggravating factors, but are not intrinsically correlated with an already established hyperplastic tendency.[26, 40]

It is suggested by Stern, Eisenbud and Klatell[28] that, if the patient has a gingival hyperplasia and if he has a history of diphenylhydantoin sodium therapy, the conclusion must not necessarily be drawn that the overgrowth was caused by the drug. The patient may have a chronic gingival hyperplasia (chronic hyperplastic gingivitis) which may simulate "Dilantin" gingival hyperplasia, but the cause must be determined. A differential diagnosis must be made and systemic conditions, such as leukemia or endocrine dysfunction, must not be overlooked. Complete systemic examination is mandatory and often a biopsy must be taken to establish the nature of the neoplasm.

■ *Clinical Appearance of "Dilantin" Gingival Hyperplasia*

Articles by Glickman and Lewitus,[40] Robinson,[27] Esterberg and White,[46] Ziskin, Stowe and Zegarelli[55] and Stern, Eisenbud and Klatell[26] report these clinical descriptions:

The typical "Dilantin" hyperplasia is painless and appears as a thickening of the gingival marginal tissue about all teeth. It is usually found in the greatest extent about the labial-gingival margin of the anterior teeth. In the uncomplicated state it is dense and with a normal color, but in the complicated state it may be deepened in color, of spongy consistency and may bleed easily.

The basic or primary lesion begins as painless, minute, discrete projections of the gingival tissue. The tissue becomes minutely lobulated, is firm, pink, resilient and does not bleed easily.

The whole gingival marginal tissue may react to the diphenylhydantoin sodium, causing the interproximal tissues to enlarge slightly and to fill the interproximal spaces, while at the same time the labial, buccal and lingual surfaces of the gingivae slightly thicken.

As this process continues, the gingival tissue thickens more and encroaches a little on the clinical crowns. The buccal and lingual gingival tissues become a linear elevation. The interproximal gingivae thicken, completely filling the interproximal spaces and begin to bulge labiolingually and buccolingually.

The gingivae then slowly enlarge and cover more and more of the clinical crowns. The interproximal tissues become mulberry-like and begin to coalesce with the lingual, labial and buccal tissues while, in the anterior part of the mouth especially, curtains of pendulant tissue are formed, causing the tooth crowns to be covered so as to reveal V-shaped enamel. In the posterior part of the mouth this can happen, but the appearance is usually a ridgelike mass along the buccal and lingual surfaces continuous with the interproximal tissue.

In a few cases, the overgrowth is so great that the crowns are completely buried by the abnormal tissue or a massive fibromatosis.[55] This is the only stage that is painful, because of food impingement during mastication, and the only stage in which the patient is inconvenienced.[56]

The end result is one of a generalized hyperplasia. The redundant tissue can be lifted away from the teeth without pain. The normal gingival attachments are unaltered, regardless of the degree of overgrowth.

During this whole process the excess tissue, if uncomplicated by inflammatory factors, remains firm, pink, resilient and does not bleed easily (similar to normal gingival tissue, except for the enlargement). It is heavily stippled and has a granular effect in all stages.

Robinson[27] and Esterberg and White[46] disagreed with the foregoing description. They observed that, in the first change from normal, the gingivae were slightly deepened in color. Ziskin, Stowe and Zegarelli[55] noted that at first the tissues were inflamed but, as the lesions enlarged, inflammation disappeared.

Regarding the qualitative and quantitative changes occurring in the gingivae of patients receiving diphenylhydantoin

sodium, Stern, Eisenbud and Klatell[26] have an interesting classification. Forty-eight per cent of their patients had no gingival reaction. In the first stage, 14 per cent had a qualitative change shown by an increased density of the gingivae exhibited by a stippling and granular effect. The second stage showed 26 per cent with a quantitative change into one or a combination of two forms: (1) a marginal festoon (which slowly increases in size, forming a roll of tissue on the crown surface); (2) an increase in size of the interdental papillae (which enlarge greatly to form triangular lobules and as they enlarge they grow toward each other with cleft formation). The third stage demonstrated 10 per cent with an increase in size in which there was an encroachment on the clinical crown. The fourth stage showed 2 per cent with so great an increase in size that there was interference with masticating function. From this classification it is obvious that the average "Dilantin" gingival reaction was mild and the severe reaction was exceptional.

This description has been of the clinical appearance of the uncomplicated "Dilantin" gingival hyperplasia. When the hyperplasia is complicated by systemic or local factors, the classic picture may be changed. Local irritants (especially poor hygiene, supragingival and subgingival calculus, improperly designed cervical margins of restorations and unfilled cervical cavities) may cause inflammation superimposed on the original hyperplastic lesion, (see photograph), thus the color, size, form and consistency of the basic hyperplasia are altered.[40]

The color[40] may change from a normal coral pink to a brilliant red with an infective marginal gingivitis superimposed. With chronic irritants, such as calculus, a venous stasis may result with bleeding into the tissues and the color will be a light blue, changing finally into a dusky blue. With conservative periodontal treatment, the abnormal gingival color will return to normal.

The size of the lesion varies greatly in different mouths and even in the same mouth, from minute abnormality to enormous overgrowth.[40] The individual predisposition to "Dilantin" hyperplasia seems to determine the degree of excess growth. It appears that, usually where the irritational factors are the greatest, the size of the hyperplasia is the greatest.

The hyperplasia is usually in the largest amount on the labial portion of the anterior teeth; relatively few cases show lingual involvement.[46]

The size of the basic lesion may increase or decrease spontaneously during diphenylhydantoin sodium therapy. With the removal of local irritations, inflammatory edema will disappear and the overgrowth may diminish until only the basic hyperplasia remains.[40]

Esterberg and White[46] noted that partial dentures, with anterior teeth abutting directly on the ridge, caused hyperplasia, the same as that which surrounded the natural teeth; also, there was overgrowth onto facings of fixed bridges. Stern, Eisenbud and Klatell[26] reported a patient who presented two pairs of small hyperplastic nodules on the palate. While the patient continued with diphenylhydantoin sodium therapy, the lesions were excised, but they returned. Only these two reports mention that "Dilantin"

hyperplasia is not entirely periodontal in character. All except Esterberg and White[46] report no hyperplasia in edentulous areas. Aside from the report of a palatal lesion, no other tissue in the body is known to react to the use of diphenylhydantoin sodium.[46]

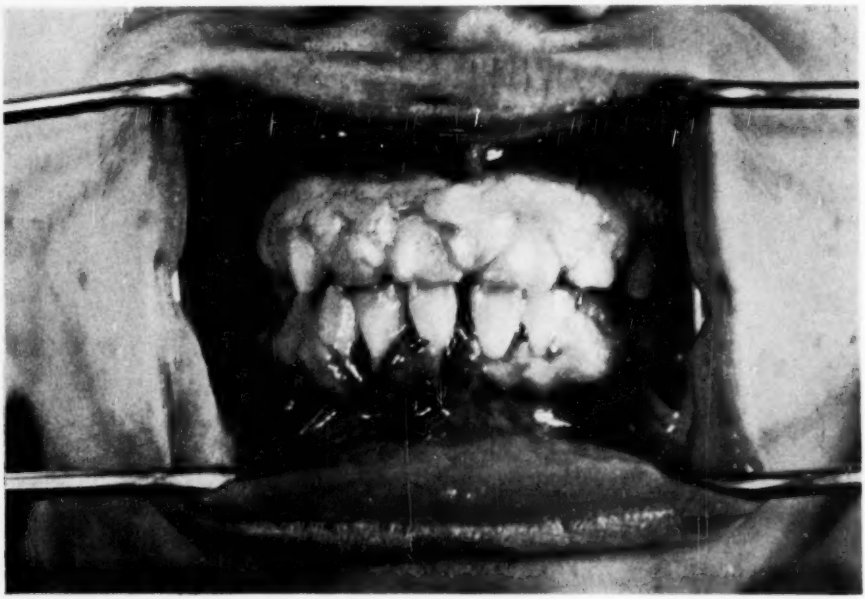
Where the teeth are separated the lesions tend to be smaller than where the teeth are in contact, perhaps because such areas are more self cleansing.[40]

Robinson[27] is the only investigator who reported that there was no hyperplasia in areas where pyorrheic lesions existed.

Ziskin, Stowe and Zegarelli[55] believed that in the absence of local periodontal treatment the "Dilantin" gingivitis progresses from mild to severe, with eventual movement of teeth and resorption of interseptal bone, producing looseness of the teeth and finally loss of the teeth. Swinehart[57] presented a case showing progressive malocclusion associated with diphenylhydantoin sodium therapy.

The form,[40] or contour, of the gingival tissue, depends on the degree of

57. Swinehart, D. R., Case Report: Progressive Malocclusion Associated with Dilantin Therapy. *Am. J. Orthodont. & Oral Surg.* 33:534 (Aug.) 1947.



Gingival hyperplasia resulting from diphenylhydantoin sodium used to control epileptic seizures of the petit mal type in a 13 year old boy. Maxillary gingivae present typical fibrous appearance for uncomplicated hyperplasia. Mandibular gingivae show typical inflammatory appearance for hyperplasia complicated by local factors (in this case oral sepsis and calcular deposit)

overgrowth and edema. With local irritations, the consistency or density may change from the normal dense, firm, stippled tissue to a soft, spongy, smooth and shiny, friable tissue.

As the final stage is reached, the overgrowth becomes so great that it is difficult for the patient to maintain any semblance of hygiene.[48] The flaps of tissue harbor fermented detritus, and conditions are favorable for the formation of subgingival calculus.

■ *Microscopic Appearance of "Dilantin" Gingival Hyperplasia*

EPITHELIUM—There is little alteration in the keratin layer. Considerable proliferative change is seen in the basal layer projecting into the underlying stroma. This occurs in long strands or epithelial extensions into the connective tissue corium called rete peg formation. Since the epithelial pegs divide and split at their ends, they may form, in cross section, epithelial whorls or pearls. There also is a change in the prickle cell layers where a slight increase is seen in mitotic figures.[55, 40, 46]

CONNECTIVE TISSUE—The proliferation is extreme; Ziskin, Stowe and Zegarelli[55] believed it to be especially great, thus accounting mainly for the gingival enlargement. There is a large, rapid formation of fibroblast or collagen bundles. There is evidence of considerable inflammatory reaction, varying in amount in different specimens, depending on the degree of inflammation present in the section at the time of biopsy. The blood vessels increase in size and numbers as do the lymph channels. The inflammatory exudate is considerable and many leukocytes and some plasma cells are seen. Edema is noted in some sections but not in others. Calcified masses of bone have been seen in the corium.[58]

Glickman and Lewitus[40] noticed that in the newly recurrent lesion there was an absence of cellular infiltration in the presence of a definite proliferative change as contrasted with a large leukocytic infiltration in the advanced lesion. They believed that this suggests the secondary nature of the inflammatory response (response to local irritations in relation to the initial hyperplastic tendency).

Esterberg and White[46] transferred bits of hyperplastic tissue directly into the anterior chamber of guinea pigs' eyes in order to determine the benign or malignant character of the growth. This method of investigation was used with both new and recurrent cases. At the end of six weeks the transplants had disappeared completely and it was concluded that the "Dilantin" hyperplastic tissue was not malignant.

■ *Methods of Treatment of "Dilantin" Gingival Hyperplasia*

Since "Dilantin" has become the drug most efficacious in the control of epileptic seizures and since gingival hyperplasia often accompanies the use of this drug, the degree of hyperplasia must be kept at a minimum, because frequently the overgrowth causes considerable annoyance if neglected; that is, painful mastication and ugly gingival appearance.

58. Zegarelli, E. V., and Ziskin, D. E., Bone Heteroplasia in Case of Dilantin Hyperplastic Gingivitis. *Am. J. Orthodont. & Oral Surg. (Oral Surg. Sec.)* 29:152 (Mar.) 1943.

The most successful method of treatment has proved to be the orthodox conservative periodontal treatment, which consists of establishing and maintaining good periodontal health.[26, 40, 46, 55, 59]

Remove all supragingival and subgingival calculus and insist upon maintenance of good oral hygiene by the patient with examinations and conservative periodontal treatment as often as the individual requires. Also, frequent and vigorous gingival massage by the patient has proved to be indispensable in controlling the amount of hyperplasia. This can be accomplished by the use of the toothbrush and the many special gingival stimulators.

Good occlusal function should be established as completely as possible by the removal of useless teeth, restoration of carious teeth, relief of occlusal trauma and restoration of edentulous areas.

Through this procedure, the marginal gingival inflammation caused by local factors (especially calculus, oral sepsis and incorrect cervical margins of restorations) will be kept at a minimum, and thus the hyperplasia will be kept at a minimum. The hyperplasia that might remain probably would be on a constitutional basis and possibly would be so mild as to cause no inconvenience. It must be forcibly stressed that, if the conservative treatment is to be successful, the patient's complete cooperation must be had at all times.

It is suggested that this procedure be instituted before or at the beginning of the therapeutic use of diphenylhydantoin sodium.[40, 55, 56]

Some investigators[40] believe that these measures are effective in relieving the inflammatory complications, but are of little or no avail in limiting the amount of hyperplasia or stopping its growth; all, however, do agree that with conservative prophylactic measures the redundant tissue will be kept at a basal level not exceeded by other methods,[26] and some[55] believe that overgrowth may even be prevented by such treatment.

Surgical periodontal treatment has been used extensively to remove the abnormal gingival tissue, but its degree of success has been minute as compared to the results of conservative periodontal treatment. Gingivectomy is the most rapid method, but is of temporary value in most cases, for the lesion returns quickly, especially if the patient does not cooperate in his home treatment of proficient oral hygiene and gingival massage.[46, 55]

The only warrant for surgical excision of redundant tissue would be when the gingival tissue is so overgrown that there is pain with mastication and when the disfigurement of the patient is great; and then it is warranted only if all conservative measures have been exhausted and if no substitute therapeutic agent can be found which would adequately control seizures.[56, 55]

When surgical means are used it is suggested[46] that only the hyperplastic tissue be removed; the gingival attachment should never be disturbed because the regeneration of epithelium is so slow that recurrence of the hyperplasia is favored.

The third method of treatment is the cessation of the use of diphenylhydantoin

59. Ziskin, D. E.; Stowe, L. R., and Zegarelli, E. V., Dilantin Hyperplastic Gingivitis; Its Treatment and Prevention. *Arch. Neurol. & Psychiat.* 46:897 (Nov.) 1941.

sodium. The lesion will gradually disappear and, with readministration of the drug, the overgrowth may not reappear in a few cases. Permanent withdrawal of the drug is permissible only if a satisfactory substitute drug is found.[28]

Experimentation is being carried on with many new drugs in order to find the perfect therapeutic agent to control epileptic seizures, but trimethadione ("Tridione") is the only one that has proved useful. Trimethadione has been used with excellent success in the control of petit mal seizures,[60] but with no success in the control of grand mal seizures.

■ *Summary*

1. At the present time, diphenylhydantoin sodium is the drug of choice, in spite of its side actions, to be used in the control of epileptic seizures.

2. Most investigators report a gingival hyperplasia of high incidence as a result of diphenylhydantoin sodium therapy.

3. The incidence of "Dilantin" gingival hyperplasia seems to be determined by individual sensitivity to the drug.

4. Almost all investigators believe that the degree of gingival hyperplasia is increased by local etiologic factors.

5. The typical "Dilantin" gingival hyperplasia appears as a slight enlargement of the gingival tissue, but may progress to a giant overgrowth.

6. The most successful method of treatment has been conservative periodontal treatment which establishes and maintains good periodontal health.—2799 *West Grand Boulevard.*

60. Lennox, W. G., Newer Agents in the Treatment of Epilepsy. *J. Pediat.* 28:356 (Sept.) 1946.

■ Teamwork in Surgery

The duration of an operation should be reduced to a minimum. This can be done by developing team work, as the many tiny pauses caused by an assistant who is just slightly behind schedule due to his slow reflexes and lack of anticipation count up very appreciably. Hemorrhage causes much delay but this can be more quickly controlled by remembering that exposure is the secret of catching a bleeding vessel. Knowledge of anatomy, use of simple methods, avoidance of depending upon assistants and development of skill will help to shorten the operating time.—*Dr. Sterling Bunnell, "Surgery of the Hand." Philadelphia: J. B. Lippincott Company, p. 97.*

Peripheral and central giant cell tumors of the head region

■ *Clayton K. Mammel, D.D.S., M.S., Ann Arbor, Mich.*

Contributions to the literature on the subtect of giant cell tumors were made by investigators as early as 1818. One of the first was Sir Astley Cooper who discussed giant cell lesions with emphasis on the ever debatable question of malignancy. Lebert, in 1845, thought giant cell tumors were always benign. Paget soon after this work augmented these studies and concurred with some of the foregoing investigation, but he was of the opinion that this or a similar tumor could become malignant. Nelaton's monograph in 1860 embraces the clinical, histopathologic variations and comparisons of this neoplasm. Virchow, perhaps the most eminent pathologist of the nineteenth century, suggested that giant cell tumors may recur and with great malignant potentialities. The contemporary investigators have used much of the material supplied by these men and have supplemented the literature with some new concepts which stimulate thought in the interested reader, practitioner and student of disease.

■ *Differentiation of Basic Types*

The giant cell group of tumors has been associated with the reparative response to injury or as a granulomatous response to inflammation. Consequently, these tumors are not considered by all pathologists to be neoplasms, yet their characteristics are definitely neoplastic. The question immediately arises as to the differentiation between the so-called epulis and the true giant cell tumor. According to Geschickter[1] the term epulis is an inclusive term which embraces gingival tumors such as granulomas, angiomas, fibroangiomas and pregnancy tumors. In his discussion of some giant cell tumors, he refers to this type which occurs on the alveolus in the region of the anterior teeth. These are often associated with trauma.

The designation of the location of the giant cell tumor should be an aid in assigning the correct nomenclature to this group of lesions; for example, distinguishing between the central and peripheral tumors. The peripheral giant cell tumor has its origin in the periosteum of the osseous tissue or in the periodontium of the dental organ. Jaffe[2] has stated, "When this tumor develops independently, it may be considered as a periosteal counterpart of the central giant cell tumor." This is the tumor which has been known by various terms, probably the most common one being epulis. The central group arises from the medullary portion of the bone or the spongiosum. The former expands super-

Department of Oral Surgery, University of Michigan, Ann Arbor, Mich.

1. Geschickter, C. F., and Copeland, M. M., *Tumors of Bone*, rev. ed. New York: American Journal of Cancer, 1936.

2. Jaffe, Henry L.; Lichtenstein, Louis, and Portis, R. B., Giant Cell Tumor of Bone: Its Pathologic Appearance, Grading, Supposed Variants and Treatment. *Arch. Path.* 30:993 (Nov.) 1940.

ficially into bone and peripherally into mucous membrane. The latter with its origin deep in bone expands in all directions usually leaving the periosteum intact.

■ *Genesis and Cytomorphology*

A recent contribution to the literature by Aegerter[3] elucidates some of the basic principles in the cytomorphology of the group of tumors discussed here. Along with other contemporaries, Aegerter acknowledges the fact that only recently the several important bone lesions have been described separately. These are hyperparathyroidism as compared to the solitary bone cyst, fibrous dysplasia of bone and eosinophilic granuloma. Since the pathologic picture of all of the above-mentioned processes reflect to some degree the pattern of fibrous connective tissue stroma and numerous giant cells, they have been confused for some time. Therefore, the most logical way to develop understanding of such a similar process is to review the basic concepts of cytomorphology and genesis of cells and tissues, thus formulating theories of tissue growth which are founded upon reasonable assumptions.

It is agreed that the formation of fibrous connective tissue is a normal response both in growth and injury. This tissue is of mesenchymal origin just as is bone and cartilage. Aegerter's explanation of this process is clear. He states that the mesenchyme persists throughout life for the purpose of serving as a precursor for the regeneration of new tissue or the replacement of other tissue which lacks the potentialities of regeneration.[3] From this primordium is derived the fibroblast; also chondroblasts and osteoclasts which in turn are capable of differentiating into mature, specialized structures. The phagocytic cells as the histiocyte and other phagocytes are products of the reticulum, and the fibroblast cannot be excluded from this group since it definitely has phagocytic powers.

The multinucleated giant cells are found in many processes in different parts of the body and are thought to be a fusion of several of these phagocytes to form one large cell. The Langerhans' giant cell of tuberculosis is different in microscopic appearance due to its characteristic epitheloid pattern.

Another multinucleated cell is that of the osteoclast, the origin and function of which have been the source of everlasting debate among pathologists. Some investigators feel that the osteoclast plays a very small role in the resorption of bone while others feel that its sole function is that of a phagocyte.[3]

It is thought that some giant cells have certain characteristics which comprise particular giant cell tumors, and some writers state that it is possible to distinguish the giant cells of solitary tumors from those of hyperparathyroidism. Aegerter is of the opinion that they are identical. He also believes that if the giant cell of osteogenic sarcoma, fibrous dysplasia, hyperparathyroidism and osteo-osteoma are considered generically related the differential diagnosis of benign giant cell tumors of bone would be greatly simplified.

A monograph by Bergstrand[4] at-

3. Aegerter, Ernest E., Giant Cell Tumors of Bone. *Am. J. Path.* 23:283 (March) 1947.

4. Bergstrand, Hilding, Notes on the Genesis of Giant Cell Tumours. *Am. J. Cancer* 27:701 (Aug.) 1936.

tempts to account for the genesis of giant cell tumors on the basis of normal bone formation. He is of the opinion that giant cell tumors are neither inflammatory nor neoplastic growths. Bergstrand seems to be opposed to the belief that it is a true neoplasm by virtue of the fact that proliferation of the giant cell process gradually ceases or often "burns itself out." From the information to follow in this paper I cannot concur with this theory. This attempt to show that giant cell tumors are formed on a basis of a local bone resorptive process does seem plausible as this phenomenon does not ordinarily occur without some stimulus. Bergstrand[4] also questions the theory that giant cells are osteoclasts but feels that they are merely "unmasked bone corpuscles."

■ *Embryologic Factors*

From the records of the surgical laboratory of Johns Hopkins hospital over a period of 35 years, 22 cases of giant cell tumors of the head and associated regions were studied. There were 2 cases of tumors of the temporal fossa, 6 of the maxilla and 14 of the mandible. In the regions of the skull developed purely by membranous formation not a single case was recorded. The regions where tumors were found are in part developed by membranous process but in the regions are also centers of cartilaginous ossification. Geschickter and Copeland[4] showed that the lesions occurring in the lower jaw were in centers of intracartilaginous ossification for the mandible. The two lesions of the temporal region were associated with the sphenoid bone which has a cartilaginous origin. In the mandible the regions concerned with tumor formation were in close proximity with that of the symphysis where Meckel's cartilage influences bone formation. Others were in the ramus where a similar situation exists.

The predominant location for giant cell tumors is the epiphysis of the long bones. Most of the studies available show that this process is initiated in regions where bone is formed from cartilage and related to the normal resorptive process of bone by osteoclasts in the histogenesis of these bones. This is further substantiated by the fact that the bones of the skull formed by membranous anlage are seldom involved by giant cell tumors or bone cysts.

■ *Possible Etiologic Factors*

The question arises as to what type of stimulus may initiate cell activity to such an extent as to form a giant cell tumor. Surely trauma is an all important condition in the histories of those who present with symptoms. Trauma may result from severe stimuli such as a blow in an athletic contest, automobile traumatism, or from manipulations about the mouth and jaws in the removal of teeth. This factor should not be minimized as a true etiologic possibility since there is definite alteration in the vascularity of the cortical bone after trauma. This is the observation of Copeland after a study of giant cell lesions of the long bones.[5] Surely this evidence can be applied to lesions of the jaws and associated regions. After such a stimulus there is a subsequent imbalance in the osteoblastic and osteoclastic action due to the interruption of

5. Geschickter, C. F., and Copeland, M. M.: Tumors of the Giant Cell Group. *Arch. of Surg.* 21:145 (July) 1930.

the periosteal blood supply. After the periosteal blood supply is interfered with the bone depends upon the medullary structure for its nourishment. This can occur only after the osteoclasts of the medullary bone have opened up new vascular channels.[3] The equilibrium of the cortical bone and medullary bone is disturbed and, consequently, the cortical bone undergoes necrosis. The compensatory proliferation of the osteoclasts of the medullary portion of the bone is at its zenith, therefore, a condition exists which resembles that of a giant cell tumor.

In enumerating the etiologic possibilities of the giant cell series those lesions which resemble the true giant cell tumor, histologically, clinically and roentgenographically but are formed on different etiological bases, should not be overlooked. Among those are bone cysts and the lesions of hyperparathyroidism. The latter present a disseminated pathologic problem of von Recklinghausen's disease or osteitis fibrosa. This is usually corrected by the removal of a parathyroid adenoma. Some investigators feel that osteitis fibrosa is not an entity *per se*, but a response to healing in a region of bone destruction. Additional evidence substantiates the fact that this tumor, which is a resultant of hyperparathyroidism, should not be classified with the other giant cell group. Others are of the opinion that there is no morphologic difference between osteitis fibrosa and other giant cell tumors.

A response simulating a giant cell tumor might occur in a region of hemorrhage, without antecedent trauma, where a phagocytic pattern is characteristic or in localized regions of calcium resorption with endothelial proliferation.

■ *Age of Incidence*

The true giant cell tumor may manifest itself at practically any age, but it is most frequently seen between the ages of 20 and 30 years. In the case of epulis this seldom occurs before the age of five years. This is best explained by Geschickter and Copeland.[5] The epulis, a peripheral lesion, is of periosteal origin. It occurs with less frequency in posterior regions about the molar teeth but more often towards the anterior portion of the mouth. It is seen most frequently in children or young adults. This is based upon the theory that the deciduous dental organ is circumvented by a periosteum with great potentialities for osteoblastic or odontoclastic action. The age of incidence of rarely under five years is, therefore, due to the fact that there is little odontoclastic action before that time.

■ *Clinical Considerations*

As stated previously, the central giant cell tumor is one which has its origin in the medullary portion of the bone, while the peripheral giant cell tumor is associated with the periosteum or the periodontal membrane. The term "epulis" is becoming obsolete since it does not identify the exact origin nor does it describe the clinical appearance. In its stead the term "peripheral" is being used. The peripheral giant cell tumor is usually situated on the facial aspect of the gingivae. It may be lobulated, highly vascular and tend to hemorrhage upon slight irritation (Fig. 1). This is the tumor frequently confused with stomatitis gravidarum or pyogenic granuloma. It is usually painless and appears with about the same frequency in the maxilla and mandible. Many clinicians

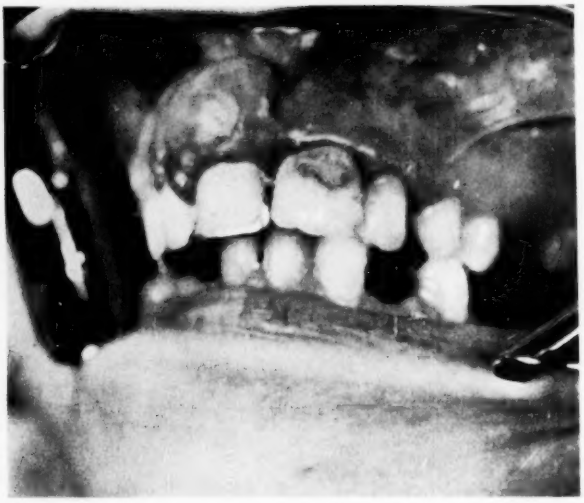


Fig. 1.—Slightly lobulated, highly vascular peripheral type tumor, situated on the anterior aspect of the gingival mucosa. (Courtesy of Department of Oral Surgery, University of Michigan, Ann Arbor, Mich.)

are not fully aware of the pathogenesis of the true central giant cell lesion. Some individuals think the process is one of true neoplasia and autonomous new growth of tissue. These tumors invade surrounding and adjacent tissues, destroy nerve trunks and bone and will recur if not adequately removed. It is thought that they may undergo malignant transformation; however, accurate record of such occurrence is vague.

The peripheral lesions, like the central giant cell tumors, are often associated with trauma. The peripheral type is not too invasive and usually causes only surface destruction and necrosis. The underlying process in the bone, provided the lesion has made sufficient advances, is one of osteomyelitis and the peripheral tumor will recur unless the underlying focus is corrected.

The central or medullary lesion is often first seen in roentgenograms or in the patient who presents facial asymmetry. The lesion is subcortical, destroying spongiosum and cortical bone and leaving only the periosteum. This group of tumors may not break through the cortical plate and mucous membrane into the mouth until far advanced (Fig. 2). Their development is usually insiduous with few symptoms; however, there may be accompanying paresthesia or anesthesia, and teeth in the affected region may become loosened or exfoliated. Solitary tumors of giant cell variety usually exhibit less intense proliferation and destruction than those of the disseminated type in the long bones.[6] There is often some associated cervical lymphadenopathy. This is not due to a metastatic phenomenon, but

6. Berger, Adolph, Solitary Central Giant Cell Tumor of the Jaw Bones. *J.O.S.* 5:154 (April) 1947.

merely compensatory inflammatory response.

■ *Roentgenographic Characteristics*

Strange as it may seem, many neoplastic processes present a very similar roentgenographic appearance. The radiologist has learned by experience not to make the diagnosis by roentgenograms alone, but there are particular peculiarities of some bone neoplasms which are distinguishing. In the case of giant cell tumors, however, there is no set of roentgenographic observations that should give a positive diagnosis. In the case of central giant cell lesions the process will probably be advanced before roentgenograms are made, consequently the defect will be one of considerable size. For the most part there is represented rarefaction in the center of the tumor mass. Toward the periphery of the lesion there is an increase in density and trabeculation of bone and a gradual blending in with the normal osseous markings. Occasionally septa may be noted as prominent projections throughout the defect. In the event that teeth are involved there is displacement of the roots with resorption and the lamina dura is not visible. There is a decided absence of

Fig. 2.—Clinical appearance of the central giant cell tumor after it has eroded the cortex and established a communication with the oral cavity. This lesion was excochleated on several occasions and within a week the mass returned with greater destructive force and with a more intense proliferation of tumor substance. After many procedures and after the loss of a great portion of the body of the mandible the growth of the lesion subsided and the defect regenerated. At different times during the treatment of the case, the surgeons were inclined to differ with the pathologist as to the true nature of this lesion, clinically it appeared as a sarcoma, but the pathologist reassured the surgeon that the lesion would "eventually burn itself out." The proliferation ceased with the suddenness with which it had arisen. (Courtesy of Department of Oral Surgery, University of Michigan, Ann Arbor, Mich.)

periosteal new bone formation over the expanded cortex.[2] As has been elicited, this appearance is not constant. In the maxilla there is often the appearance of irregular osteolytic foci. It may have a slightly moth-eaten pattern as is characteristic of osteomyelitis. In the mandible it is most likely to take on cystic tendencies with an irregular periphery and coarse trabeculations. The septa already spoken of are not always visible if the film is overexposed. The lesions of central giant cell tumors, although extremely destructive, remain more or less encapsulated with an elevation of the periosteum and with cavitations and decreased density of the surrounding cortical bone. This type of lesion may be differentiated from a true cyst by a lobulated effect and by scalloped or undulating margins.

■ *Histologic Characteristics with Variances*

In a discussion of the histology of these tumors, other processes must be recognized and considered due to the similarity in histologic architecture; namely, von Recklinghausen's disease, Paget's disease, sarcoma, fibrous dysplasia of bone, ossifying fibroma and bone cyst. Only a few can briefly be considered here.

FIBROUS DYSPLASIA—In this process is evidenced a young fibrous connective tissue stroma with proliferation of young matrix. There is present immature masses of vascular tufts and some attempts at bone formation. In giant cell tumors there is almost no attempt at new bone formation.

Giant cells are sometimes seen but are not present in large numbers, and by some workers it is thought that they never exist in fibrous dysplasia.

VON RECKLINGHAUSEN'S DISEASE OF BONE—This is also composed of aggregates of giant cells in a fibrous connective tissue stroma; however, if there is proliferation of the stroma the diagnosis is difficult.

OSSIFYING FIBROMA—The stroma is again similar to that of fibrous dysplasia. There are more attempts at the formation of mature bone, and the activity of the fibrous connective tissue stroma is neither so active nor is its vascularity so great. A diagnosis based entirely upon the slide material of fibrous dysplasia and ossifying fibromas is almost impossible.

OSTEOGENIC SARCOMA—The osteoblasts from the inner layer of the periosteum undergo neoplastic proliferation. They make attempts to form bone, but as the process gains momentum, the attempts made vaguely resemble bone formation. They resemble giant cell tumors of bone very closely and consequently it is difficult to differentiate sarcoma from a malignant form of giant cell tumor.[3] Methods by which microscopic differentiation may be made will be discussed in subsequent paragraphs. One feature is that of osteoid formation. The mother cell produces fibrous connective tissue while the osteoblasts produce bone. From this it may be concluded that the two processes may overlap one another depending upon the stage of the process observed. Although there are other processes similarly related they are not within the scope of this paper and the entities considered may be mentioned only briefly.

GIANT CELL TUMORS (CENTRAL)—Osteoclasis which occurs is thought by some to be a normal physiologic response. In abnormal situations the osteoclasts are found in large numbers; for example, in large regions of hemorrhage. The concept that osteoclasts themselves possess neoplastic potentialities is debatable, since the cell itself is in adult form. The microscopic picture of giant cell tumors is not one in which there is a proliferation or mitosis of cells themselves but merely their presence is accounted for as a response to the neoplastic process. They are the result of a coalescence of some smaller microphages.

The matrix of this tumor being fibrous connective tissue is no different than that of the other similar tumors which have a fibroblastic stroma. The diagnosis of giant cell tumors is based upon the fact that the stroma itself is of a neoplastic nature. If the stroma is one of numerous new vascular channels and young fibroblasts, this is a fairly accurate index of the growing potentialities the tumor possesses. The characteristics cannot be based on the appearance of the giant cells.

The giant cells found in a variety of the lesions mentioned are for the most part identical. They are produced primarily for the purpose of phagocytosis. They should not be used solely in the diagnosis of giant cell tumors of bone. The fibrous matrix is also used as a criterion for establishing a diagnosis for these tumors. The histopathologic diagnosis of giant cell tumors of bone is founded on the neoplasticity of the fibrous matrix and its immaturity.

Frequently there may be seen a tumor with abundant giant cells containing many nuclei. The large cells are engulfed in a cellular stroma which contains many lymphocytes which may resemble the nuclei of the giant cell. Geschickter and Copeland[3] predicate that giant cell formation is a resultant of hyperplasia of osteoclasts. In the stroma spindle cells may be seen, some small vascular channels and not too infrequently hemorrhage. The lymphocytes of the stroma seem to be related to the giant cells, while the spindle cells are related to the fibrosing osteitis and the hemorrhagic regions to the vascular channels. The large giant cells are not too unlike osteoblasts.

Lichtenstein and Jaffe[2] consider the giant cell tumor as a neoplasm of a specific type which arises from the undifferentiated connective tissue of the marrow. Much of the description of this tumor has been based upon the multinucleated giant cell but great attention should be given to the stromal elements on which the potentialities of this lesion are really based. This has been previously discussed. These workers feel the word "benign" has been misused, thus giving a false impression of the tumor, for often this process becomes extremely aggressive and locally invasive with various metaplasias of the stromal elements.

The over-all microscopic appearance of this lesion has invasive characteristics. The stroma exhibiting young spindle cells, giant cells and lymphocytes is said to be more representative of the rapidly growing lesions. These cells have a small amount of cytoplasm with abundant chromatin and a large nucleus. Spindle cells are present in large numbers, and are oval in contour with a fairly well demarcated nucleus and the cytoplasm of these cells is minimal. When the giant cells

Fig. 3.—In this section is clearly shown the type of giant cells present in the central giant cell lesion. There are present several aggregates of large multinucleated cells surrounded by a fibrous connective tissue pattern. No atypical mitosis is seen. Although this section does not elicit all the types of giant cell patterns, it does show the cellular tendencies characteristic of this group of tumors. (Pathologic material from the University of Michigan, School of Medicine, Department of Pathology, Ann Arbor, Mich.)

are present in large numbers adjacent to regions of hemorrhage the question may arise as to whether these cells are phagocytosing the blood pigment or whether they are products of a neoplastic process. A modification of these giant cells is seen in osteogenic sarcoma. However, the cell found in this sarcomatous process is one which has a more vesicular type nucleus and not so many nuclei as has the benign type or foreign body giant cell. In a malignant lesion, mitosis may be observed.[7]

Grading of these tumors by virtue of cytologic structure has been attempted by Jaffe and his associates.[2] This system of grading is founded upon the aggressiveness of the stromal elements, the most benign types being grade I. In this grade there is evidenced no atypism of the stromal components, and the cells are spindle in contour, while in the advanced types they take on polygonal forms. In grade I, the tumors show little evidence of new bone formation; however, at the periphery of the lesions this architecture may be modified. In grade II, the stromal elements are vast and closely connected often lending themselves to a whirled configuration. The fibroblasts are apt to be more oval and perhaps contain two or more vesicular nuclei and much chromatin. According to Jaffe there is almost a total lack of spontaneous ossification in this group.

7. Thoma, K. H., *Oral Pathology*, 2nd ed. St. Louis: C. V. Mosby Company, 1944.

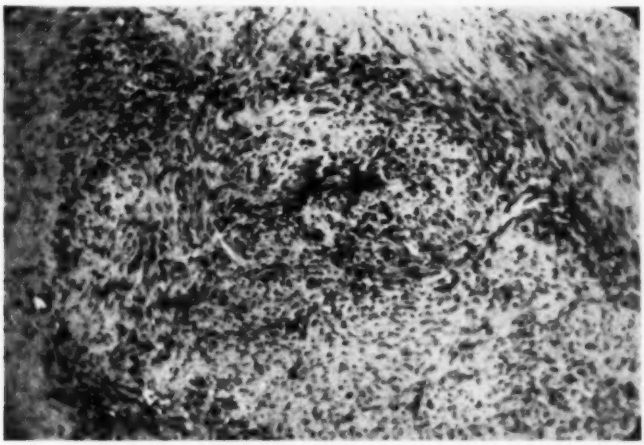


Fig. 4.—Here is shown the tendency toward a whorled appearance of the stromal elements. The fibroblasts are more mature in type toward the center of the mass and more vesicular in type toward the periphery of the lesion. Also at the periphery the stroma is not nearly so dense. In the center of the section may be seen an excellent example of the formation of the large multinucleated giant cell. One feature often found in the giant cells of these tumors is the large numbers of nuclei present. Here is shown the formation of a cell which will have approximately 18 nuclei when formation is complete. (Pathologic material from the University of Michigan, School of Medicine, Department of Pathology, Ann Arbor, Mich.)

The prognosis of grade II is strongly for recurrence. The grade III group contains malignant characteristics, sarcomatous type of stroma and metastatic potentialities. The giant cells are dwarfed and possess fewer nuclei. The malignant variants of giant cell tumors are not agreed upon by all investigators. Coley[8] feels that those lesions which later become malignant were improperly diagnosed in the beginning. In a series of cases taken from Johns Hopkins hospital eight of which are supposedly the most illuminating were examined by competent pathologists. The cases which had undergone malignant changes and metastasis were found not to have been originally bona fide examples of benign tumors. In no instance has the explicit transformation of a giant cell lesion into a sarcoma been proved. Coley accounts for transformation of allegedly benign tumors into malignant growths on the basis of a lesion being unsuccessfully treated and subsequently traumatized, thus presenting a fecundate site for the development of osteogenic sarcoma.

■ *Differential Microscopic Diagnosis*

Many diagnostic errors result in interpreting healing responses after pri-

8. Coley, William B., Malignant Changes in the So-Called Benign Giant-Cell Tumor. *Am. J. Surg.* 28:768 (June) 1935

mary operation as malignant change. In cases of infection there might be present pyknosis or cloudy swelling of cytoplasm and increased proliferation of spindle cells with vesicular type nucleus. The characteristics of the giant cells in the nonmalignant giant cell tumor are the presence of many nuclei, often more than 15 per giant cell.[1] This is not characteristic of malignant tumors. Associated with this are polymorphonuclear leukocytes and round cell infiltration with alternate areas of hemorrhage, engulfed with a loose fibrous stroma. This cellular pattern closely resembles osteogenic sarcoma, but making an accurate diagnosis is difficult. In this variant there is a less cellular stroma and the giant cells are larger and more numerous than those seen in sarcoma. The differential diagnosis of these processes is best made on the basis of the new bone formation present.

Figures 3, 4 and 5 are photomicrographs showing some of the cellular architecture of the peripheral and central giant cell lesions.

■ *Summary and Conclusions*

From the foregoing sections of central and peripheral giant cell tumors, although

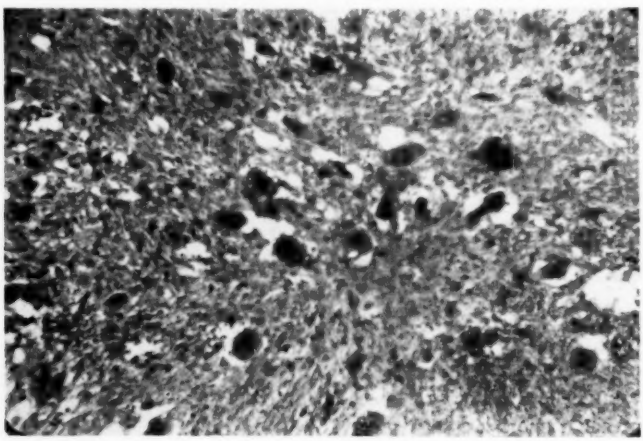


Fig. 5.—The similarity of the peripheral and central giant cell lesions as to histology is evident in a great many instances. This section is from a peripheral type tumor, and exhibits many of the features present in the central group of tumors. There are numerous giant cells with more or less cytoplasm than seen in the previous sections, and the fibroblasts, for all practical purposes, are of approximately the same maturity. If this lesion were to recur the cellular architecture would undoubtedly undergo metaplasia, which is characteristic of giant cell tumors. The giant cells would in all probability become a less prominent feature while there would be an increased rate of proliferation of the underlying fibrous connective tissue stroma. This, of course, is what leads one to suspect sarcomatous change. (Pathologic material from the University of Michigan, School of Medicine, Department of Pathology, Ann Arbor, Mich.)

not complete, perhaps can be derived a better understanding of the histopathology of this group of tumors.

1. Giant cell tumors of the skull and associated regions are usually correlated with the distribution of the centers of ossification which have cartilage as its precursor.

2. The formation of giant cell tumors, either peripheral or central, is usually initiated by some stimulus, such as trauma, with subsequent alteration in the vascularity of the periosteum.

3. The giant cells present in these tumors are usually of the osteoblastic type and possess as many as 15 to 25 nuclei. The giant cell is similar to the one found in several lesions of bone.

4. The diagnosis of giant cell tumors should be made on the basis of the activity and the maturity of the fibrous connective tissue stroma and not on the giant cells alone.

5. When these lesions recur with an immature, highly proliferative stroma, there is no positive evidence that they undergo sarcomatous change.

6. A spindle cell variant of giant cell tumor may exist which resembles a sarcoma.

7. The term "epulis" should be abandoned. In its stead the term "peripheral" giant cell tumor should be used to designate those lesions arising from the periosteum or the periodontium, while "central" giant cell tumors should embrace those lesions arising in the spongiosum or medullary portion of bone.

■ Registration of Microscopes

In a recent issue of "The Laboratory" the announcement of a registry service available to owners of microscopes was made. This service is made possible by The Scientific Apparatus Makers of America, a national association. This society is offering the service to all owners of microscopes in an attempt to help them curtail the theft of such instruments. With the increase in demand for microscopes during the last few years, and the increase in the cost of producing these instruments, increasing numbers of thefts have been reported. Most microscopes carry a serial number on the microscope support arm. In order to register a microscope it is necessary to write a letter to The Scientific Apparatus Makers of America at 20 Wacker Drive, Chicago 6, Illinois, requesting a registration card. The association will forward a card for each individual microscope. It is not possible to register groups of microscopes.

If a registered microscope is stolen the local police should be notified immediately. The registration number and any other distinguishing features of the microscope should be forwarded to the association headquarters in Chicago. The association will notify the authorities in the larger cities where such instruments are most likely to be sold. When the instrument is found the association should be immediately informed so that an adjustment can be made in its records.

A list of stolen instruments can be obtained by interested persons free of charge upon request. —*"Microscopes Should Be Registered," The Laboratory, 17:124.*

Differential diagnosis of tumors of the neck

■ *Conrad J. Baumgartner, M.D., Beverly Hills, Calif.*

A high percentage of patients with tumors of the neck, particularly of the lymph nodes, are referred by physicians to dentists for the removal of dental sepsis. Unfortunately, in most instances, the nodule in the neck has nothing to do with dental sepsis. Either a primary growth is found, or after a careful investigation it is determined that a silent primary carcinoma has been present in the oral cavity, but has been overlooked. The fact that dentists are so frequently the intermediary between the physician and the surgeon prompts a discussion of the clinical features of some of the tumors of the neck.

■ *History and Physical Examination*

It is of the utmost importance to get a careful history of the patient who presents himself with a lump in the neck, and to follow this with a systematic examination of the neck.

The history must include not only the duration, rapidity of growth, the question of fever and pain, but also must include investigations into each possible focal source, such as nasal discharge, respiratory infection, dental sepsis and hoarseness or cough.

An examination of the neck should follow a definite pattern. With the neck in slight flexion toward the side to be examined, to avoid muscle tension, palpation should begin in the submental region and proceed to the submaxillary, subparotid, jugular, occipital and supraclavicular regions. The thyroid region should be examined next. Simple palpation of a thyroid will miss at least 50 per cent of small nodular goiters. To examine the thyroid gland properly, the neck is placed in slight forward flexion. The thumb of each hand is placed over the trachea, and the index and middle fingers of each hand are placed behind each sternomastoid muscle. Pressing forward lightly with these two fingers against the posterior lateral surface of the gland, the patient is asked to swallow. Small nodules at the lower pole can be detected easily in this manner. Any mass which moves on swallowing is probably connected with the thyroid structure, and any mass which does not move is most likely not connected with the thyroid.

■ *Enlarged Lymph Nodes*

SUPPURATIVE CERVICAL LYMPHADENITIS—Suppurative cervical lymphadenitis is characterized by an abrupt onset. The history of a sore throat or dental sepsis preceding the swelling is significant. There is tenderness and there are signs of inflammation. The so-called tonsillar lymph gland, located just below the angle of the jaw, usually is involved.

CHRONIC CERVICAL LYMPHADENITIS—Usually several glands are enlarged when chronic cervical lymphadenitis is present. To differentiate between this

Presented at the annual meeting of the Southern California Society of Oral Surgeons, Los Angeles, May 16, 1947.

Associate professor of surgery, College of Medical Evangelists, Los Angeles, California.

type, tuberculosis adenitis and Hodgkin's disease may be difficult, and is sometimes impossible without a biopsy, particularly in the absence of definite foci of infection.

TUBERCULOUS LYMPH NODES—Tuberculous lymph nodes are not as common in this country as they were before milk was pasteurized and cattle were tested for tuberculosis. Since World World II tuberculous cervical adenitis has again become prevalent in Europe. Next to cancer, the most common cause of chronic asymmetric enlargement of cervical lymph nodes is tuberculosis. Tuberculous glands are more likely to be matted together and the skin may be adherent. There is a tendency toward fistulization and the discharge may be caseous. Evidences of tuberculosis elsewhere usually are found, and skin tests are strongly positive.

HODGKIN'S DISEASE—Hodgkin's disease is characterized by progressive enlargement of lymph nodes, fever, moderate anemia and leukopenia. The nodes are discrete in contradistinction to the matted characteristics of tuberculous glands. The enlarged lymph nodes are movable and have a peculiar elastic consistency to palpation. They are usually observed on one side of the neck, and they enlarge slowly. Sooner or later glands are palpable in the axilla and groin, and roentgenograms often will reveal mediastinal nodes. A Pel-Ebstein type fever is another characteristic of Hodgkin's disease. Tenderness is not a characteristic, but suggests suppurative lymphadenitis. A differential diagnosis, however, usually is impossible without a biopsy.

LYMPHATIC LEUKEMIA—A generalized enlargement of lymph nodes accompanied by an enlarged spleen signifies lymphatic leukemia. A complete blood count will verify the diagnosis.

INFECTIOUS MONONUCLEOSIS—Infectious mononucleosis is an acute infectious disease characterized by fever and a swelling of the cervical lymph nodes. There is an abnormally large number of mononuclear leukocytes in the blood, but the number of red blood cells is normal. Diagnosis is confirmed by heterophil agglutination with sheep's blood.

SYPHILIS—A generalized lymphadenopathy, lymphocytosis and eosinophilia with normal white and red blood cells suggests syphilis. It is imperative that a blood Wassermann be performed on all cases of lymphadenopathy. A complete blood count is necessary also.

■ *Midline Lesions*

Lymphadenitis at or near the midline is rare. Any swelling in this region, with the exception of the superficial sebaceous cyst, usually is connected with the thyroid structure.

THYROGLOSSAL CYST AND FISTULA—In embryonic life the anlage for the thyroid begins at the foramen cecum at the base of the tongue. From here it travels as a tube (thyroglossal duct) forward to the hyoid, then it travels down in front of the trachea to form the thyroid. Ordinarily this tube atrophies, but if it persists a cyst or fistula will result. The cyst is unilocular, and, although it may occur anywhere along the course of the embry-

onic thyroglossal duct, it usually presents itself at, or a little below, the hyoid bone. As with all thyroid structures, it moves on swallowing. The cyst is not tender unless it is secondarily infected. The fistula may be primary or it may occur after incision and drainage of the thyroglossal cyst. The discharge is of a tenacious ropey consistency. Often a pencil-like tract can be felt beneath the skin which extends to the hyoid bone and moves on swallowing.

THYROID GLAND TUMORS—The thyroid should be examined in the manner previously described. The size of the thyroid can be determined in this manner, and small nodules which are readily overlooked can be detected only by this procedure. Any nodule in the region of the thyroid which moves on swallowing is most assuredly a part of the thyroid structure. Conversely any nodule which does not move on swallowing is most likely not thyroid structure.

■ *Movable Single Lateral Tumors of the Neck*

The laterally located tumors of the neck which are single and movable are branchial cysts, carotid body tumors, single lateral aberrant thyroid, cystic hygroma and neurofibroma.

BRANCHIAL CYST—A branchial cyst is always unilocular and it has a characteristic position in relation to the anterior border of the sternomastoid muscle. The cyst may occur at any level, but it is usually below the level of the bifurcation of the carotid artery. Although it is below the cervical fascia it is always anterior to the carotid sheath and it tends to bulge outward. If the cyst occurs in connection with a fistula, the fistula also has a constant relation to the anterior border of the sternomastoid muscle. Aspiration of the contents of the fistula will frequently reveal a shimmering material which may contain cholesterin crystals.

CAROTID BODY TUMOR—Only between 300 and 400 cases of carotid body tumor have been reported. These tumors arise from the carotid body which is located in the carotid notch. The carotid notch is at a level with the upper border of the thyroid cartilage. The tumors are, therefore, at a higher level than branchial cysts. In Lahey's[1] series of 18, he noted that when they enlarged, they did so upward under the angle of the jaw, and usually bulged inward rather than outward. Because they are attached to the carotid arteries, these tumors may be moved laterally, but they cannot be moved up and down. Aspiration is indicated if the diagnosis between branchial cyst and carotid body tumor is doubtful, as extirpation of a carotid body tumor is a procedure which is associated with a rather forbidding mortality.

LATERAL ABERRANT THYROID—Lateral aberrant thyroid tumors may be multiple, but often a single discrete nodule is presented. Although they may occur anywhere in the neck, they frequently occur along the jugular vein. Lahey stated that they do not tend to bulge upward under the jaw as do carotid body tumors. Lateral aberrant thyroid tumors frequently are

1. Lahey, F. H., and Warren, Kenneth, Tumors of the Carotid Body. *Surg., Gynec. & Obst.* **85**:281 (Sept.) 1947.

associated with nodules in the ipsilateral side of the thyroid. A discrete movable tumor accompanying a nodule within the thyroid gland is at least suggestive of lateral aberrant thyroid.

CYSTIC HYGROMA—Cystic hygroma tumors are caused by embryologic sequestration of lymphatic vessels from the main network. Although classified here under single tumors, they are always multilocular. They are noticed first in infancy. They are usually situated in the lower part of the neck, frequently in the supraclavicular region. However, the cystic hygroma tumors may invade not only the neck but also the axilla, chest, mediastinum and the arm. Cystic hygroma is translucent and the aspirated fluid is straw colored, except when it is discolored brown from intracystic hemorrhage.

NEUROFIBROMA—Neurofibroma tumors occasionally occur along the course of some of the larger nerve trunks of the neck. There are no distinguishing features. They become large in the early stages and they can be moved laterally to the long axis of the nerve.

■ *Silent Metastatic Nodule*

In a series of 1,867 patients with primary cancer of the mouth and pharynx, Martin and Morfit[2] reported that metastasis was the first symptom in 145, or 7.8 per cent. Although the primary growth is within the mouth, it is to be expected that almost 10 per cent of patients will not be aware of the growth in the mouth, and their first attention will be drawn to the nodule within the neck.

The neck nodule occurs frequently as a single discrete nodule which almost invariably will be hard and partially fixed. A history of nosebleed, sore throat or tongue, or hoarseness may give a further clue for diagnosis. Such a nodule in the neck warrants a meticulous examination of tongue, tonsils, nasopharynx and larynx in search of a primary cancer.

Those nodules occurring in the lower cervical region are most frequently associated with a thyroid malignant growth or they may be secondary to a malignant growth below the clavicle. The "Virchow's node," or "sentinal node," occurring in the supraclavicular region, was formerly associated with advanced carcinoma of the stomach. It is now known to occur with any malignant lesion in the chest or the abdominal cavity or even as far distant a source as carcinoma of the prostate.

■ *Summary*

Patients with cervical lymphadenopathy or with cervical nodules frequently are referred to dentists for the removal of foci of infection. Such nodules frequently have no association with any focus of infection. A knowledge of the clinical aspects of some of these lesions may save the dentist from sharing an embarrassing situation with the referring physician.

2. Martin, Hayes, and Morfit, H. M., Cervical Lymph Node Metastasis as the First Symptom of Cancer. *Surg., Gynec. & Obst.* 78:133 (Feb.) 1944.

Bilateral osteotomy of the rami for agnathic mandible: *report of case*

■ *Douglas F. Pincock, D.D.S., Ogden, Utah*

A 22 year old white man was referred to the dental clinic at Hoff General Hospital August 17, 1945, for correction of an agnathic mandible. He complained of protrusion of the upper anterior teeth and the inability to bite on his front teeth. He gave a history of sucking his thumb until he was about seven years old.

The patient had had no serious illnesses or operations. His father and mother were both living and well. There was no tuberculosis, insanity, epilepsy or diabetes in the family.

EXAMINATION—Examination revealed a well developed, well nourished, white man. Heart and lungs normal; pulse 76; blood pressure 80/120; white blood cells 8,200; red blood cells 5,100,000; hemoglobin 11.6; Kahn negative; urine clear, specific gravity, 1.020; sugar negative; no pus cells.

Oral hygiene was poor. All four of the first molars were missing. The arches were narrow and constricted, with a 2 cm. overbite. There was extensive caries throughout the mouth with periodontoclasia, generalized and moderate. There was extreme protrusion of the upper teeth with retrusion of the lower teeth and an underdeveloped chin (Fig. 1, *A, B* and *C*).

TREATMENT AND COURSE—Pyorrhea treatments were given this patient during the month of September 1944, and all the carious teeth were filled. The lower anterior teeth were extracted on October 8. There was no temporary replacement of the extracted teeth. Orthodonic bands and arch wire appliances were made and cemented to the upper and lower posterior teeth. These appliances were later utilized with intermaxillary rubber band traction in repositioning the mandible. The patient was sent to surgery for a surgical reconstruction of the mandible on October 28, under nitrous oxide-oxygen and ether anesthesia.

An incision was made down the anterior border of the right ramus, extending from the coronoid process to the retromolar triangle. The periosteum and submucous tissues were dissected laterally and medially, exposing the middle third of the ramus. The mandibular nerve was observed and retracted by placing the medial retractor above its entrance into the mandibular foramen. The lateral retractor was then positioned, exposing the ramus. A Luc motor bone saw was used to cut through the ramus. The cut was made superior to the mandibular foramen parallel to the occlusal plane of the lower teeth and directed downward in a lateral direction. The mucous membrane flaps were closed with black silk sutures. This procedure was repeated on the left ramus. The mandible was then free and could be moved forward in a more favorable position.

Later in the day when the patient had recovered from the anesthetic, intermaxillary rubber bands were applied, pulling the mandible forward. This immobilizing traction was maintained for two months.

The postoperative course was uneventful.

There was a moderate amount of swelling in both cheeks and some soreness on swallowing. This had completely subsided after eight days. The incision healed by first intention and the sutures were left to sluff out.

The upper six anterior teeth were extracted on January 15, 1945, and a partial denture was inserted immediately. Healing was normal. A fixed bridge was inserted January 29, completing the reconstruction of this patient's mouth.

RESULT—The mandible was moved forward the width of a bicuspid. This produced a more normal intermaxillary ridge relationship and provided good functional occlusion.

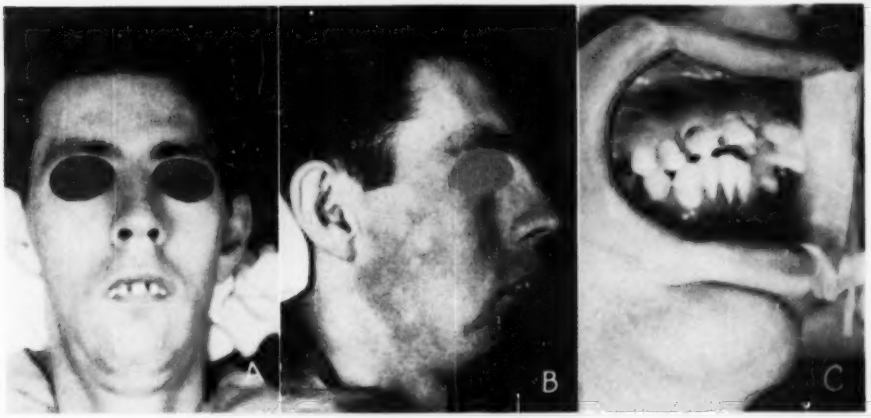


Fig. 1.—A: Front view of patient before treatment for agnathic mandible. B: Profile view showing protrusion of the upper teeth, retrusion of the lower teeth and an underdeveloped chin. C: closeup of patient's teeth

Fig. 2.—Patient after treatment. His appearance was greatly improved

The fragments healed in good position and there was no limitation of motion.

The appearance of the patient was improved (Fig. 2, *A*, *B* and C). He developed more confidence and a friendlier personality. When the patient first came to the clinic it was practically impossible to get him to talk. He would only answer direct questions. After correction, he became much more affable and had friends throughout the hospital.

CONCLUSION—This case is presented to substantiate the contention that favorable psychologic changes result after the improvement of a deformity.

The intraoral technic employed in this case has the advantage of not leaving any external scars and avoids the hazard of injury to the facial nerve. Continuity of the mandibular nerve is conserved with this procedure. The operation is done in a contaminated field, which would increase the possibility of postoperative infection. However, considering the large number of surgical procedures that are done in the mouth and the few postoperative infections, this hazard is minimal.

This same technic can be used in the reconstruction of prognathism. I have done four such cases, all with good results.

Care must be taken in both types of cases to provide strong anchorage to the teeth and adequate intermaxillary immobilization to position and hold fragments in the desired relationship.—*First Security Bank Building.*

Foreign body in tongue:
report of case

■ *Thomas J. Hagen, D.D.S., Fort Gulick, Canal Zone*

A 24 year old veteran, admitted to the Oakland Regional Hospital, Oakland, Calif., July 30, 1945, stated that on June 2, 1944, he was wounded by a gunshot while in combat in Italy. The bullet entered the right cheek at the level of the right mandible and traversed the oral cavity up to the left maxillary antrum, making its exit through the left temple. He made a satisfactory recovery from this injury and was medically discharged from the army. His chief complaint on admission to this hospital was fulness and swelling of the right side of the tongue, recurrent during the past six months. There was frequent drainage from a small fistula on the right lateral surface of the tongue.

EXAMINATION—Examination of the head revealed a small scar on the right side of the face about 2 cm. superior to the inferior border of the mandible in the region of the second molar. There was a small scar in the left temporal area.

Oral examination showed the right molars to be missing. The upper left second and third molars were missing. The tongue appeared appreciably swollen, and there was a moderately enlarged mass palpable in the middle third of the right side of the tongue. At the right lateral border of the tongue there was a small fistula, from which was draining a slight amount of purulent material.

Dental roentgenograms revealed no remaining roots or pathologic condition in the

Lieutenant colonel, U.S. Army, now stationed with 368th Station Hospital, Fort Gulick, Canal Zone.

Lateral roentgenogram showing foreign body in the tongue. Insert: Occlusal view showing foreign body in the tongue

edentulous regions. A lateral roentgenogram (Illustration) of the tongue revealed what appeared to be the crown of a tooth embedded in the right side of the middle portion of the tongue, and medial to the tooth there was a small fragment.

COURSE AND TREATMENT—The patient was started on a course of penicillin, 30,000 units every four hours, beginning July 31. He was considered ready for surgical intervention August 5. On August 6 he was premedicated with 1½ grains pentobarbital sodium and 1/150 grain scopolamine hydrobromide, and under endotracheal ether nasal tube, the two foreign bodies were removed. To reduce hemorrhage, 1 cc. epinephrine 1:10,000 was injected in the region of the incision. The tongue was held out with a towel clip and a 1.5 cm. incision was made over the right lateral surface of the tongue. The incision was deepened and enlarged by blunt dissection, and the foreign body, which proved to be the crown of a lower molar, was easily enucleated. The other small particle was located and removed. The wound was closed with two deep sutures. The following day the tongue appeared in excellent condition. The sutures were removed the fifth day. The wound continued to heal by first intention, and the patient made an uneventful recovery.

Acute lymphatic leukemia with a fatal termination: *report of case*

■ *Arthur R. Dewey, D.D.S., Ventura, Calif.*

Leukemia may be defined as an acute or a chronic disease of unknown etiology, involving principally the blood-forming organs and characterized by widespread disorderly prolification of the white cells and their precursors in the various organs and tissues of the body. It manifests itself by the appearance of abnormal leukocytes in the peripheral blood. Invariably the disease has a fatal termination.[1]

Leukemia may occur in either an acute or a chronic form. The three chief variations are: myelogenous leukemia, characterized by an increase in myelocytes and myeloblasts in the blood stream; lymphatic leukemia, character-

1. Saghirian, L. M., and Jones, C. A., Acute Monocytic Leucemia. *Am. J. Orthodont. & Oral Surg.* 28: Sec. Oral Surg. 561 (Oct.) 1942.

ized by an increase in lymphoblasts or lymphocytes; monocytic leukemia, characterized by an overproduction of monocytes in the blood stream.

Difficulty in diagnosis of the type of leukemia often arises from improper identification of the leukocytes, particularly in the early stages of development.[2]

The oral manifestations in all types of leukemia are outstanding because of their early onset and the danger that results from surgical intervention, which, in the early stages of the disease may be undertaken because the true condition has not been recognized.

Local hemorrhage, ulcerations, gingival hyperplasia and fetor oris, which, according to Thoma,[3] have been termed leukemia stomatitis, are the signs commonly seen first by the oral surgeon.

Lassitude, progressing pallor, loss of weight, gastrointestinal disturbances, pain in bones, ecchymosis of the skin or oral cavity, enlargement of the liver and spleen and local or generalized lymphadenitis are also symptoms commonly found. The oral surgeon should, therefore, inquire into the history of the patient, study the blood count and clotting time and refer the patient to an internist for careful investigation of the general condition.

I recently had occasion to handle a case of lymphatic leukemia and I report it because the usual oral and generalized symptoms were not apparent, and because of the extreme violence and rapidity of the termination.

A 29 year old white woman, employed as a secretary, was first seen on the afternoon of August 22, 1946. She complained of a severe odontalgia of four days' duration.

EXAMINATION—Examination revealed a well developed, well nourished young woman, evidently in acute pain. She stated that for the past four or five months she had been feeling "tired and run down" and had suffered intermittent pain in the involved teeth. For the past four days the pain had been so excruciating she had been unable to report for work and had been waiting for it to subside as it always had in the past.

Oral examination revealed an apparently normal mouth, well restored and with good oral hygiene, except for advanced caries of the right maxillary second bicuspid tooth and the left mandibular second bicuspid tooth. These two teeth had been used as bridge abutments in the past, but recurring caries had necessitated removal of the restorations which had never been replaced. Intraoral roentgenograms of the involved teeth revealed direct carious exposure of the pulps of both teeth but no apical pathologic condition was apparent.

TREATMENT AND COURSE—Under a local anesthesia of 2 per cent procaine hydrochloride, both teeth were removed without undue trauma. There was no evidence of hemorrhage and the patient was discharged from the office.

On the evening of August 26, four days postoperatively, the patient called stating that on the previous day she had developed a temperature and a sore throat, and that at the time of the call, she was feeling quite ill. The patient was in a toxic condition and was evidently acutely ill. Examination revealed a temperature of 103 F., pulse 112 and respiration 28.

The patient was admitted at once to the hospital where examination of the mouth revealed a normal postoperative picture of healing sockets, a grayish membrane covering the gingivae and a coated tongue. The breath was fetid and there was a slight swelling of the neck and a distinct submaxillary lymphadenopathy.

The throat was fiery red and there was edema of the soft palate and anterior pillars. The posterior pillars were spotted with a grayish white membrane.

Physical examination by a resident physician was essentially negative with the exception of moist rales at the base of the left lung and a rapid heart rate. Small subcutaneous hemorrhages of the legs were noted, which the patient said were from striking her desk at work. The condition was diagnosed tentatively as acute pharyngitis due to streptococcus.

2. Resch, C. A., Oral Manifestations of Leucemia. *Am. J. Orthodont. & Oral Surg.* **26**: Sec. Oral Surg. 901 (Sept.) 1940.

3. Thoma, K. H., *Oral Pathology*, ed. 2. St. Louis: C. V. Mosby Company, 1944.

The patient was given intramuscular injections of penicillin, 40,000 units every three hours, and continuous hot saline throat irrigations. A complete blood picture and urinalysis were ordered.

On August 27, the first blood report was: hemoglobin 11.9 Gm.; red blood cells 2,840,000; white blood cells 107,200; bleeding and clotting time normal.

The differential count showed 89 per cent to be undifferentiated mononuclear leukocytes, most immature cells were small lymphocytes and there were a few polymorphonuclear leukocytes. A working diagnosis of acute lymphatic leukemia was made.

Medical consultation was called and the opinion of the consulting physicians was that the outcome would be fatal.

An internist was asked to supervise medical treatment. The temperature was 104.6 F., pulse 126 and respiration 42 at this time and intensive therapy was instituted. Oxygen was administered, ice bags were placed on the great veins and the amount of penicillin was increased to 100,000 units every two hours. A transfusion of 350 cc. of whole blood and 200 cc. of 5 per cent glucose was given.

August 28. The patient seemed much worse. The spleen was palpable to the level of the iliac crest. The chest bilaterally was full of rales and the lower lobe of the left lung was dull to percussion. The white blood count had increased to 266,000. It was doubtful if the patient would live through the night.

August 29. The patient was very restless and irrational and it was necessary to employ narcotics to keep her quiet. Five hundred units of plasma were given. The white blood count was 782,000.

August 30. The general condition of the patient seemed to be improved but she was so irrational the outlook remained very poor. Five hundred cubic centimeters of whole blood were given. The temperature remained between 103 and 104 F.

At the request of the patient's sister in Columbus, Ohio, a telephone consultation was held with Dr. Charles Austin Doane, professor of medicine at the Ohio State Medical School. From the history Dr. Doane believed that this case came within his classification of lympholeukosarcoma, a lymphosarcoma which invades the blood stream rather than the tissues. He stated that a few of his patients responded to roentgen ray over the spleen.

August 31. The patient's general condition remained the same. During the afternoon she was given 50 roentgen units of high voltage roentgen therapy over the spleen.

September 1. The patient suddenly expired at 4:10 a.m.

AUTOPSY—Pertinent findings at the postmortem examination were: congestion of both lungs; heart enlarged to the midaxillary line; myocardium thickened to 1 inch at its greatest portion and ⅝ inch at its least; liver huge, filling the abdomen to the level of the fifth rib, in the anterior axillary line down to the umbilicus and weighing 8 pounds; kidneys and spleen greatly enlarged.

The gross anatomic diagnosis was (1) generalized lymphadenopathy; (2) pulmonary edema; (3) pneumonia, bronchial, terminal; (4) fibrinous pericarditis; (5) cardiac hyperplasia; (6) passive congestion of the liver with hepatomegaly; (7) splenomegaly; (8) renomegaly and (9) adrenal insufficiency. The immediate cause of death was circulatory collapse.

DISCUSSION—Although the occurrence and removal of two infected teeth marked the beginning of a precipitous downhill course, the leukemia had been present for many months, as was noted by the advanced pathologic condition of the vital organs at autopsy. It was becoming more severe before the dental infection appeared. The clinical and roentgen evidence of the dentition was such that one would expect a complaint as presented by the patient. The dental pathology must, therefore, be considered as unrelated to the leukemia, and the extractions likewise must be considered unrelated to the systemic disease.

Barnfield[4] has reported that a relation exists between dental procedure and leukemia in

4. Barnfield, W. F., Leukemia and Dental Procedures. *Am. J. Orthodont. & Oral Surg.* 31: Sec. Oral Surg. 329 (June) 1945.

approximately 1 case in 12. The leukemia becomes more acute in approximately half of the patients who undergo dental operations. However, dental procedures performed on patients with leukemia may be unrelated to the systemic disease. Then because signs of leukemia are sometimes misinterpreted as being of dental origin, dental procedures are performed. Two possible relations of leukemia to dental procedures are (1) lowered tissue resistance to bacterial invasions because of leukemia and (2) leukocytic infiltration of the dental pulp may cause toothache in patients with leukemia.

Dr. Doane's suggestion of lympho-leukosarcoma is explained by MacCallum[5] as a term to designate those cases in which a definite tumor is developed in some organ or tissue, which is composed of lymphoid cells which seem to escape into the blood stream giving rise to leukemic alterations of the blood. Others refuse to recognize this as anything distinct from lymphoid leukemia in which there may be extensive tumorlike infiltrations of the tissues.

In the case reported here, the only systemic complaint of the patient was that of a tired and weak feeling. Her oral condition aroused no suspicion, and the pathologic condition of the two teeth involved was certainly enough to warrant her complaint of pain. There was no doubt but that she had a severe case of chronic leukemia and would have expired in a short time without surgical intervention. However, the leukemia must have become exacerbated after the dental operation. It is imperative for each oral surgeon to inquire into all systemic complaints of the patients however slight these complaints may be.—2427 *East Main St.*

5. MacCallum, W. G., *A Textbook of Pathology*, ed. 4, Philadelphia: W. B. Saunders Company, 1928.

Osteomyelitis of the mandible treated with aureomycin: *report of case*

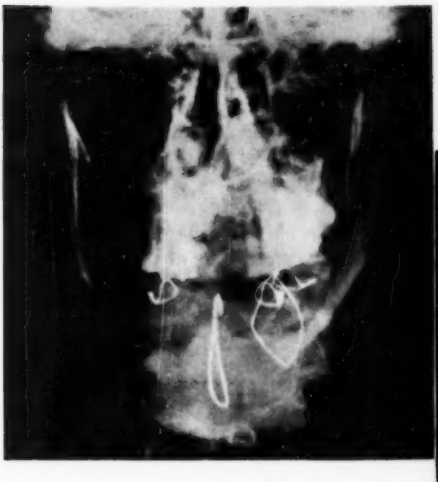
■ *George F. Egan, D.M.D., New York*

Since the introduction of aureomycin, there has been no recording of its use in the treatment of osteomyelitis. Aureomycin possesses bacteriostatic and bacteriologic activity against numerous gram-positive and gram-negative bacteria. Oral administration of aureomycin to mice protected them against infections produced by streptococcus hemalyticus beta in experiments by a group of doctors from Johns Hopkins University School of Medicine, Baltimore.[1]

HISTORY—A 54 year old white man was admitted to the hospital in January 1948, with a fracture of the mandible and cellulitis of the face and neck. Ten years before, he had been admitted for osteomyelitis of the tibia with resultant amputation of the leg. At that time he had osteomyelitis of the mandible, and was also treated for syphilis.

TREATMENT AND COURSE—When seen in January 1948, his edentulous mandible was fractured on the right side with a 3 cm. loss of bony substance. Circumferential wiring of the fractured parts of the mandible to the patient's lower denture was the treatment of choice (illustration, left).

1. Bryer, Morton S.; Schoenbach, E. B.; Chandler, Caroline, A.; Bliss, Eleanor A., and Long, Perrin H., Aureomycin: Experimental and Clinical Investigations, *J.A.M.A.* 138:117 (Sept. 11) 1948.



Left: Roentgenogram showing wiring of the fractured parts of the mandible to the patient's lower denture

Right: Roentgenogram showing "melting away" of the mandible, which resembled many sequestrums

The result was satisfactory from a functional standpoint; however, drainage continued from the osteomyelitis process in spite of 250,000 units of penicillin twice a day. In June 1948, the wires had to be removed because of bone degeneration at the point of contact of the wire with the bone.

In August, penicillin was discontinued and aureomycin (20 mg. intramuscularly daily) was administered. The discharge of pus was reduced dramatically, and at the end of four weeks the external wound had closed and healed. The degenerative process of the bone apparently was arrested.

In September 1948, after the supply of aureomycin was unavailable, a new lesion on the left side of the face appeared, following a spontaneous fracture of the left side of the mandible. Because aureomycin was not available, 250,000 units of penicillin twice a day was reinstituted with no improvement. The lesion increased in size, both in the soft tissue and in the bone. Drainage was profuse.

A roentgenogram showed a "melting away" of the mandible, which took on the appearance of many sequestrums resulting from pathologic fracture (illustration, right). Cultures revealed staphylococcus albus, streptococcus hemalyticus beta and aerobacter aerogenes.

On January 5, 1949, the patient was admitted to the hospital and aureomycin was again instituted (3 Gm. daily by mouth) with a remarkable diminution in quantity of pus after 48 hours.

After 12 days a culture showed only streptococcus hemalyticus beta and a 90 per cent reduction of pus. Blood serology was negative throughout this period. Blood chemistry was within normal limits, except the level of phosphorus, 4.6. The edema subsided and permitted surgical intervention for the removal of small sequestra and granulations. The laboratory report on these specimens revealed no distinct pathologic features aside from chronic osteomyelitis.

On his eighth postoperative day the patient was dismissed from the hospital with a minimal discharge from the wound. Subsequent check-ups revealed no further discharge, no edema and no pain.

COMMENTS—This case had failed to respond to the usual regimes of therapy and surgical intervention, and the prognosis for saving the mandible was extremely poor. Perhaps the tertiary syphilis had caused arteritis with resultant avascularity and bone necrosis. The dissolution of bone was proceeding at an alarming rate and resection of the jaw seemed inevitable. The dramatic improvement after the first aureomycin administration was viewed with considerable question, but the results after the second administration of aureomycin were even more convincing. In both instances, after administration of the drug, the copious flow of pus was reduced to a minimum in two weeks.

SUMMARY--1. A case of chronic osteomyelitis of the mandible is presented.

2. The response to aureomycin was remarkable in view of the patient's age, debility and the extent of the infection.—525 *East 68th Street.*

■ Tissue Reaction to Plastics

Tissue reactivity to FM-1 nylon, cellulois, lucite, cellophane and teflon was determined by intraperioneal implantation in experimental animals. All except the last-named substance produced proliferative foreign body reaction. Teflon, an inert, semiflexible material cannot be wet with water and is nonadherent. Clinical application of this unusual substance in surgical procedures is mentioned. —*H. H. LeVeen and J. R. Barberio, "Tissue Reaction to Plastics Used in Surgery With Special Reference to Teflon," Ann. Surg. 129:74, 1949.*

VOLUME 8 • JANUARY 1950 • NUMBER 1

The practice of oral surgery

CURRENTLY THERE IS great concern among many of the practitioners of oral surgery throughout the country in regard to the future of our specialty. Some of the men in this field have become extremely apprehensive regarding their personal future and also the future of the specialty of oral surgery.

It is felt by many that we are in danger of losing our identity as oral surgeons in the true sense of the word and of becoming relegated to the service of extraction of teeth alone. This sense of insecurity arises apparently from what is thought to be inroads into the field of oral surgery by some of the allied medical specialties. It is alleged that these medical specialists, because of the change in the character of their practice since the advent of antibiotics and the sulfonamides, are looking to new fields of endeavor. It is true that the character of medical practice changes from year to year, and that some of the medical specialists are beginning to include in their field problems that were foreign to them a few years ago. A few of these problems happen to be ones that some oral surgeons feel rightfully belong within the scope of oral surgery.

The thought that all diseases of the oral cavity and adjacent structures fall within the scope of the oral surgeon and should be treated by no other specialty is not founded on good judgment. Many of these conditions are medical in their concept inasmuch as they may be related to disease or involve anatomic regions entirely outside the oral cavity. These lesions are best treated in many instances by the medical specialist. In order to give the patient the best possible service in the management of diseases of the mouth and associated structures treatment should be instituted by the specialist who is best qualified by training and experience to handle the condition, whether he be a physician or a dentist. In most instances conditions peculiar to the oral cavity are best treated by the dentally trained oral surgeon. There are, however, some medical specialists who are very well qualified to handle these conditions. Other medical specialists are attempting to qualify themselves by extensive graduate and postgraduate study in the university

laboratories and clinics, as well as by attendance at postgraduate clinics and intensive short courses of study.

The dental oral surgeon, in order to hold his own or to gain ground in the management of those problems, which by virtue of his training and experience he is best able to handle, must be a progressive individual. He must take an active part in hospital practice and in the teaching programs of the hospital. He must be attentive and active at staff conferences and staff meetings. He must keep up on the literature in his own field as well as the allied fields and be regular in attendance at postgraduate courses and conferences. He must make himself and his ability known among his colleagues by association with them in hospital life.

The dental oral surgeon who spends all of his time within the confines of his own four office walls so that he continues to see fewer and fewer of the major problems will find that these problems will be taken over by his medical colleagues.

Oral surgery as a specialty cannot be legislated into a position of respect and prominence in the existing order. The specialty must earn this position by creating an attitude of confidence and demonstrating to the medical and dental professions and to the public the superiority of qualified service by well trained oral surgeons.

With the improvement in the training of increasing numbers of young surgeons in the field of hospital oral surgery and the development of the influence of the American Society of Oral Surgeons and the American Board of Oral Surgery, there seems little to fear regarding the future of our specialty.—*Reed O. Dingman.*

VOLUME 8 • JANUARY 1950 • NUMBER 1 SURGICAL ORAL PATHOLOGY

Squamous cell carcinoma involving the periodontal membrane

■ *Joseph L. Bernier, D.D.S., M.S., and Richard W. Tiecke, D.D.S., M.S., Washington, D. C.*

While involvement of the periodontal membrane by malignant tumors is not rare, it is usually seen only in the terminal growth of such lesions. Usually the gingiva is infiltrated first by tumor cells which break through the basal cell layer of the surface epithelium into the immediate submucosa. The deeper structures are more often involved on the lingual and buccal surfaces where the invading cells quickly infiltrate the periosteum.

Primary squamous cell carcinoma is not as frequently seen on the buccal gingiva as elsewhere in the oral cavity due in part to a better developed stratum corneum which exhibits more complete maturation.[1] When it is involved, however, the prognosis is rather poor because of early invasion of the mandibular or maxillary bone.

Central squamous cell carcinoma of the mandible and maxilla is rare; however, a few such lesions have been reported. These tumors most likely arise from epithelial remnants of the developing teeth and from the epithelium of suture lines which abound, particularly in the maxilla. These tumors cause teeth to become loosened, a condition often suggestive of malignancy.

A case is reported here in which the primary lesion began in the floor of the mouth and extended up over the gingiva and then down into the intact alveolus to invade the periodontal membrane.

HISTORY—A 58 year old white man sought medical treatment because of pain in the floor of the mouth of two years' duration. Examination revealed a region of ulceration in the midline of the floor of the mouth and extending onto the lingual gingiva. The inferior surface of the tongue was also involved. There was limited and painful motion of the tongue, and palpable left submaxillary lymph nodes which clinically were believed to contain metastatic tumor. The entire dentition was intact although the teeth were badly stained and covered with heavy calculus deposits.

Roentgenograms suggested involvement of the bone in the region of the symphysis. Biopsy was performed with a resulting diagnosis of carcinoma, squamous cell.

OPERATION—Preparatory to roentgen therapy six anterior teeth were removed with some attached tissue. This was done under procaine hydrochloride anesthesia.

MICROSCOPIC—An incisor tooth to which fragments of soft tissue were adherent was sec-

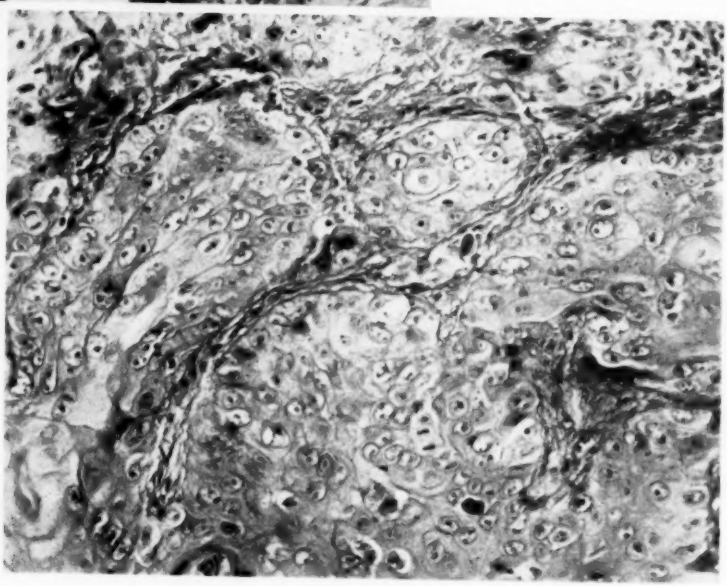
Chief, Dental and Oral Pathology Section, Armed Forces Institute of Pathology, Washington, D. C. Executive Secretary, and Pathologist, Registry of Dental and Oral Pathology of the American Dental Association.

Resident, Oral Pathology, Armed Forces Institute of Pathology, Washington, D. C.

1. Bernier, Joseph L., and Ash, J. E., *Atlas of Dental and Oral Pathology*, ed. 4. Washington, D. C.: Registry Press, 1948.

Fig. 1.—Carcinoma of the periodontal membrane. Along the righthand side of the picture the periodontal membrane has been completely replaced by tumor cells. Note the close apposition of the tumor masses to the cementum. The separation between the cementum and the dentin is artefact

Fig. 2.—Carcinoma of the periodontal membrane. A higher power of the invading tumor cells demonstrate their altered morphologic characteristics. Numerous mitoses are evident



tioned. Tumor cells were present along one surface only extending from near the gingival crevice to the root apex. From this section it appeared that the tumor extended downward from the alveolar ridge. It was composed of infiltrating strands of squamous epithelial cells showing pronounced morphologic alteration. Some keratin was noted and there were many mitotic figures. The tumor elements appear to infiltrate directly into the periodontal space, replacing the entire periodontal membrane and being in close association with the cemental surface. Some chronic inflammatory elements were also noted as well as areas of organizing hemorrhage. The cementum had been pulled away from the dentin during sectioning, creating an artefact (Fig. 1 and 2).

COMMENT—It is rather uncommon to see a squamous cell carcinoma from associated regions of the oral cavity invading the periodontal membrane and leaving the alveolar bone intact. However, this may occur more frequently than supposed since block sections are not often prepared.

A previous biopsy of other material in apposition to the same teeth failed to reveal any malignant process and was coded as chronic granulation tissue. However, this tissue was removed from the opposite side of the tooth where the tumor had not yet invaded. This case illustrates very well the need for having teeth with attached pieces of soft tissue examined by a pathologist in those cases where associated oral structures present a suspicious change not readily diagnosed by clinical methods. While involvement of the periodontal membrane is a common occurrence in malignant central tumors of bone, it is uncommon to find direct extension of a surface lesion into the periodontal space, as occurred in the case which is presented.

Hemangioma of the lip

■ *Joseph L. Bernier, D.D.S., M.S., and Richard W. Tiecke, D.D.S., M.S., Washington, D. C.*

Hemangiomas are composed of vascular channels which may vary in size from capillaries to cavernous spaces. In a simple hyperplasia of the blood vessels, such as is often seen in the granuloma pyogenicum, there is an orderly rather than a purposeless arrangement such as is noted in hemangiomas. Frequently both lymph and blood spaces are involved; however, the diagnosis of lymphangioma is not indicated unless the change primarily affects lymph channels.

Misplacement or distortion of endothelial elements during embryologic development may persist as a hamartomatous hemangioma;[1] indeed such a background may exist for most vascular tumors. In the tumor presented here such an etiology is strongly suspected. These tumors are usually small at birth but increase in size with age.[2] When there is an excessive degree of endothelial proliferation the vascular channels may be obliterated by infoldings of the vessel walls. These lesions are spoken of as sclerosing hemangiomas.

Vascular tumors are not only disfiguring, since they frequently grow to a large size, but they may complicate other procedures or be potentially malignant in themselves. They may

1. Bernier, Joseph L., and Ash, J. E., *Atlas of Dental and Oral Pathology*, ed. 4. Washington, D. C.: Registry Press, 1948.

2. Thoma, Kurt H., *Oral Pathology*. St. Louis: C. V. Mosby Company, 1944.

be located most anywhere on the face, but are particularly common on the lip or forehead where they create a cosmetic problem as they become larger. Many of these congenital malformations are considered as nevi being often referred to as "birthmarks." Most of these can easily be removed if they are small but occasionally the infiltrating type is encountered when surgical procedures become more difficult. Hemorrhage due to trauma is not an infrequent occurrence so that ulceration and pain may be common complications. Hemangiomas may occur centrally in bone and are often mistaken for other tumors or inflammatory lesions. Thoma[2] cites a case in which the tumor was located in the region of the mental foramen. Severe hemorrhage occurred following the extraction of a tooth which resulted in the death of the patient.

In spite of the tendency of some vascular lesions to invade, and the very cellular character of others, this tumor does not metastasize. There are, however, malignant counterparts of the benign hemangioma such as the hemorrhagic sarcoma of Kaposi which is always fatal.

REPORT OF CASE—An 11 year old white girl was born with a blue tumor mass on the right lower lip. This mass frequently became large and often traumatized producing considerable pain and discomfort.

Examination of the nose was negative externally. Endonasally the mechanics were good and the soft tissues healthy. The mouth revealed a large mass of hemangiomatous vessels involving the margin of the right angle of the mouth and occupying a space of about 2 cm. by 4 cm.; it was relatively superficial. Immediately behind

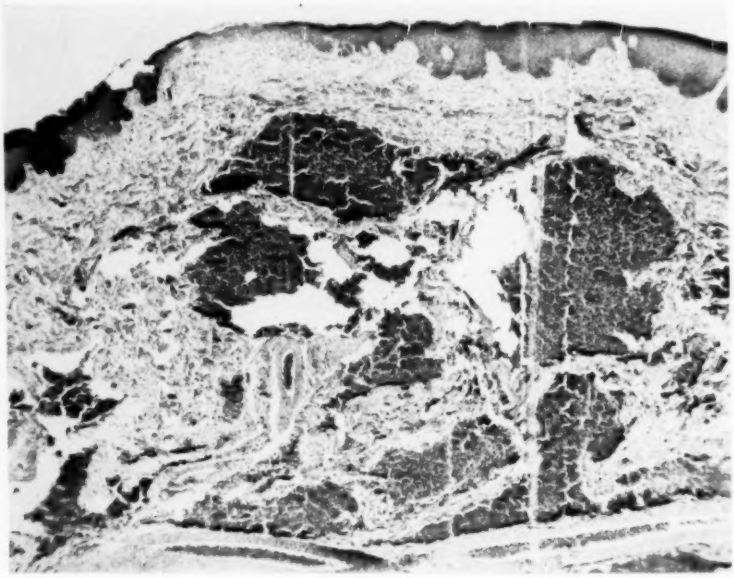


Fig. 1.—Hemangioma of the lip. Note the large vascular spaces filled with red blood cells undergoing partial organization. The epithelium covering the surface is mildly hyperplastic and shows very little keratosis

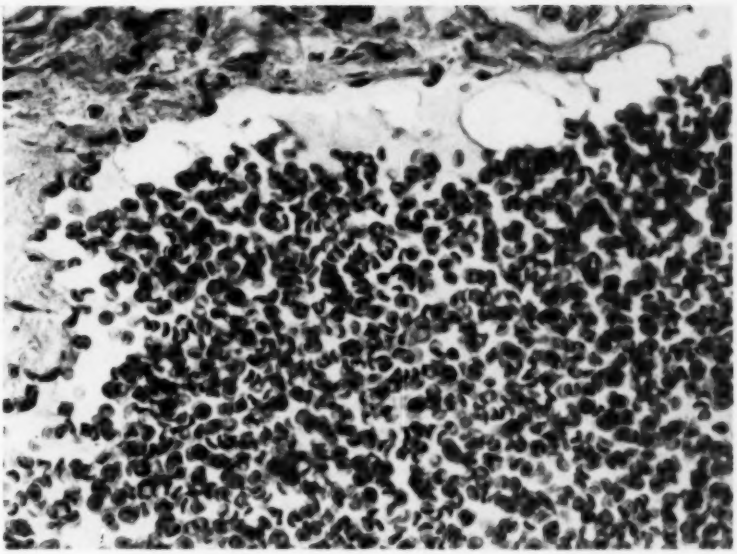


Fig. 2.—Hemangioma of the lip. A higher power of one of the large vascular channels. The space is filled with partially disintegrated erythrocytes. The irregular endothelial lining of the vascular space can be seen

this and just in front of the parotid gland there was a mass approximately 2.5 cm. in diameter and 1 cm. in thickness. It was discrete, soft and nonfluctuant.

The throat revealed that a tonsillectomy had been previously done with good results. Posterior rhinoscopy and indirect laryngoscopy were negative. The ears were negative.

OPERATION—Atropine sulphate, 1/300 grain, was given 45 minutes preoperatively. Thiopental sodium was administered as the anesthetic.

An elliptic incision was made entirely around the mass on the lip and carried down to the subcutaneous tissue. After removal the subcutaneous incision was closed with chromic catgut and the mucous membrane closed with silk.

MICROSCOPIC OBSERVATION—The tissue examined exhibited extensive formation of neoplastic blood vessels, most of which were filled with red blood cells. Photomicrographs are shown in Figures 1 and 2.

In addition there were numerous dilated lymphatic channels filled with a pale staining amorphous material. There was a spotty distribution of chronic inflammatory elements which were chiefly lymphocytes and plasma cells. A small hard structure was also noted which appeared to be a salivary stone.

COMMENTS—This tumor in all probability should have been removed earlier when it was smaller in view of the tendency of hemangiomas to become larger with age. Disfigurement such as that produced by this lesion may well have resulted in personality changes.

Plasmocytoma

■ *Joseph L. Bernier, D.D.S., M.S., and Richard W. Tiecke, D.D.S., M.S., Washington, D. C.*

The plasmocytoma is a fairly rare tumor, relatively few having been reported to date. Claiborn and Ferris[1] reviewed the literature previous to 1931 and found only 12 cases to which they added two of their own. Cappell[2] reported two cases in a group of 122 oral tumors of all types. New and Childrey[3] noted two cases in a review of several hundred malignant neoplasms of the palate, nasopharynx and tonsil. Mattick and Thibaudeau[4] reported 19 cases of plasma cell tumors of the upper respiratory tract and added one of their own. Figi, Broders and Havens[5] reported on 11 cases of plasma cell myeloma, all of which were from the upper respiratory tract.

Plasmocytomas are generally considered to be of the extraosseous origin,[6] being more common in the upper respiratory tract, with the nasal cavity and nasopharynx the usual sites.[7] They have also been reported in the tonsil, pharynx, palate, tongue, floor of the mouth and maxilla. According to most observers it is more common in the man than in the woman and most frequent in the 30 to 70 year age group.

Fiji and associates conclude that on the basis of their experiences, plasmocytomas are malignant lesions and should be classified, however, this is not a universally accepted concept. According to Stout and Kenney[6] the lesion is probably far less fatal than multiple myeloma, but is unquestionably potentially malignant as it is often locally invasive.

The tumor is composed of plasma cells in a massed formation and supported by a reticular stroma of connective tissue for which reason it has often been referred to as a unicellular neoplasm. The plasma cells present the usual "cartwheel" appearance of the nuclei, few mitoses and usually numerous Russell-Fucsin bodies. Of great importance is the fact that the plasma cells often exhibit very little morphologic alteration.

It is agreed by most investigators that plasma cells are of reticulo-endothelial origin and that their function in inflammation may be to stimulate the production of immune globulins necessary for healing.

Although other lesions such as chronic ulcers, polypi and certain granulomas may contain plasma cells, the quantity of cells noted in the plasmocytoma makes differentiation easily possible. It is the presence of masses of these cells unassociated with inflammation that is the important diagnostic criterion.

REPORT OF CASE—The patient was a 59 year old white man. In June, 1948, he sought medical aid because of nasal obstruction on the left side which had been present for seven years. There had never been any severe pain and there was no history of nasal discharge. His main complaint was difficulty in breathing.

Upon examination a large swelling was noted in the left anterior triangle of the neck, which the patient said had been diagnosed clinically as a cyst 15 years before and treated by roentgen therapy. Anterior to this large swelling some discrete lymph nodes were palpable. The left nasal cavity revealed an obstruc-

1. Claiborn, L. N., and Ferris, H. W., Plasma Cell Tumors of the Nasal and Nasopharyngeal Mucosa. *Arch. Surg.* 23:477 (Sept.) 1931.

2. Cappell, D. F., The Pathology of Nasopharyngeal Tumours. *J. Laryng. & Otol.* 53:558 (Sept.) 1938.

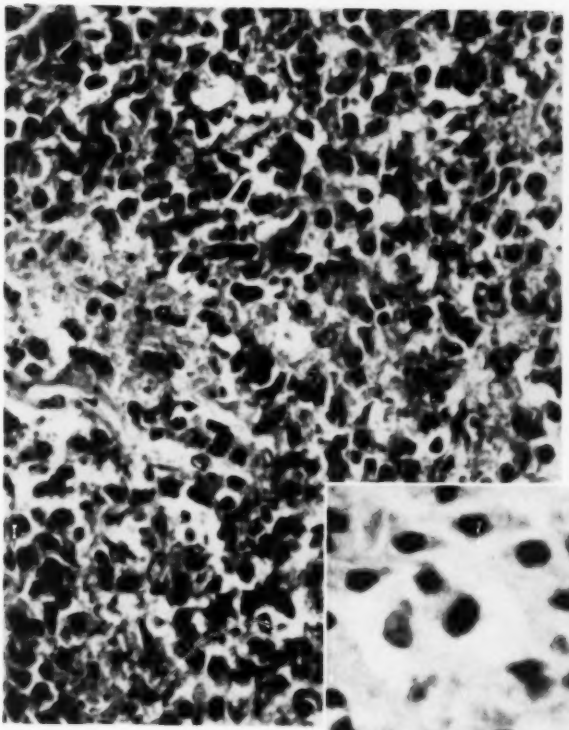
3. New, G. B., and Childrey, J. H., Tumors of the Tonsil and Pharynx (357 Cases). III. Malignant Tumors Exclusive of Adenocarcinoma of the Mixed Tumor Type (220 Cases). *Arch. Otolaryng.* 14:713 (Dec.) 1931.

4. Mattick, W. L., and Thibaudeau, A. A., Extramedullary Plasma-Cell Tumors of the Upper Air Passages; with Report of a Case. *Am. J. Cancer* 23:513 (March) 1935.

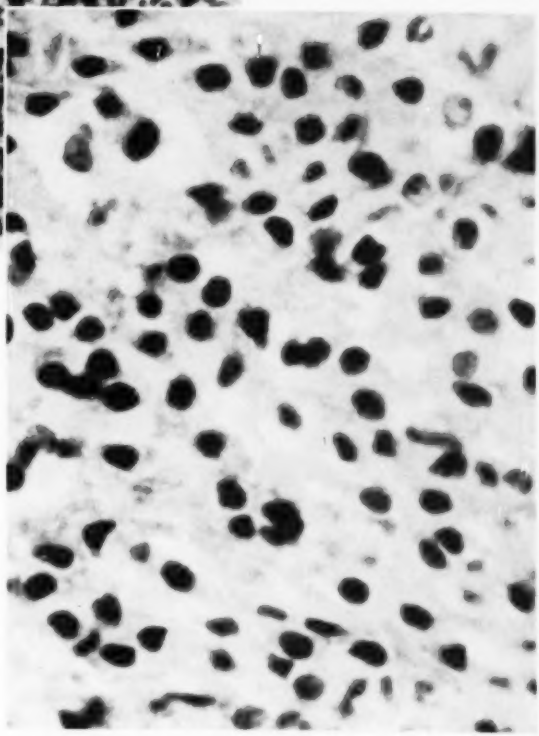
5. Figi, F. A.; Broders, A. C., and Havens, F. Z., Plasma Cell Tumors of the Upper Part of the Respiratory Tract. *Ann. Otol., Rhin. & Laryng.* 54:283 (June) 1945.

6. Stout, Arthur Purdy, and Kenney, Francis R., *Cancer*, 2:261 (March) 1949.

7. Bernier, Joseph L., and Ash, J. E., *Atlas of Dental and Oral Pathology*, ed. 4. Washington, D. C.: Registry Press, 1948.



Left: The entire field shown contains plasma cells which are hyperchromatic. Only occasional cells demonstrate the "cartwheel" nucleus characteristic of the plasma cell. Occasional mitosis is evident



Right: A higher power of the same field shows more clearly the character of the plasma cell. In the upper portion of the picture, lighter staining cells show the chromatin distribution within the nucleus

tive growth which was diagnosed clinically as a polypoid degeneration of the middle turbinate.

A roentgenogram of the region disclosed a filling defect of the left antrum. On the basis of this roentgenogram and a biopsy, a diagnosis was made of "papillary adenoma, probable low-grade carcinoma."

OPERATION—On August 8, 1948, a Caldwell-Luc approach was made. The external bony wall of the maxillary antrum was eroded and thin. There were three small holes extending through to the periosteum. The canine fossa was easily broken through and a fairly firm tissue mass was removed. The lining of the antrum was thin, peeling away with the tumor, leaving a clean, white, bony cavity. Near the anterior mesial angle the tissue was more adherent, necessitating removal with a curet. An antral window was made and the incision was closed without drainage.

FOLLOW-UP—Convalescence was uneventful and there was no sign of recurrence nine months after operative procedures. However, during this period one of the lymph nodes in the neck became larger.

MICROSCOPIC OBSERVATION—The gross material consisted of irregular pieces of firm white and light brown tissue aggregating a mass approximately 4 cm. in diameter.

The cellular morphology was fairly uniform consisting of plasma cells with abundant eosin-staining cytoplasm. The cell borders were ill defined in some places, their irregularity suggesting branching. These plasma cells were present in considerable numbers exhibiting the "cartwheel" arrangement of the nuclei. Under high power there was considerable debris lying in between the cells as well as reticulum fibrils. Fibroblasts, some lymphocytes and epithelial cells were present in small numbers. Vascularity was moderate in some areas and very prominent in others (see illustrations).

COMMENTS—The term plasmocytoma should be reserved for only those tumors composed of true plasma cells unassociated with inflammation.

The most common neoplasm from which they must be differentiated is the multiple myeloma of bone. This is extremely important since the latter lesion carries a serious connotation.

■ BIOPSY

An unqualified diagnosis of cancer even in advanced cases can be made only by the pathologist. Lacking microscopic tissue examination, a diagnosis can be no more than "tentative" or "clinical." It is the responsibility of the examining physician to determine whether or not the lesion clinically looks enough like cancer to warrant the taking of a specimen for biopsy.—*Hayes Martin, "Cancer of the Head and Neck," American Cancer Society, Inc.*

■ Metastatic Lesions of the Mandible

Q.—How common are metastatic lesions of the mandible?—*C. Litton.*

A.—Metastatic carcinoma of the jaw is rare. Carcinoma of the thyroid, prostate and breast produce the most frequent osseous metastasis. Rare examples of metastasis to the mandible from carcinoma of the lung, kidney and rectum have been reported.

■ Treatment for Ameloblastomas

Q.—Should all ameloblastomas be treated by resection of the part involved?—*D. Cooley.*

A.—No. If there is no break through the cortical plate or invasion of the periosteum a conservative approach should be taken. The mass should be completely removed and a sclerosing agent used in the cavity. If there is a recurrence, or if there is involvement of the periosteum, then more radical procedures are indicated. These include local or hemiresection of the mandible.

■ Angina Pectoris Complication

Q.—What would be the anesthetic agent of choice when the patient requiring extraction of teeth has a history of angina pectoris?

A.—The use of any general anesthetic agent that might produce anoxia might seriously embarrass the physiology of the heart muscle. A weak general anesthetic agent such as nitrous oxide probably would fail to give complete anesthesia without seriously cutting down the oxygen content of the blood. An anesthetic of this type would be definitely contraindicated. If it became definitely necessary to anesthetize the patient with a general anesthetic agent, cyclopropane would probably be the drug of choice. This drug produces a pleasant, quick induction and moderate relaxation with immediate recovery. It has the advantage of being a potent anesthetic agent in the presence of high oxygen concentrations. Its use is contraindicated in patients with cardiac irregularities but is perfectly safe when properly used in patients with other cardiac and circulatory disorders.

The safest method of managing a patient with the history as stated would be premedication with 1½ to 3 grains of pentobarbital sodium and infiltration or block anesthesia with procaine containing minimal amounts of vasoconstricting agents.

■ Action of Penicillin

Q.—What is the mode of action of penicillin?

A.—It is believed at the present time that penicillin in some way interferes with the nutritional phase in the development of bacteria. The dosage of the penicillin determines whether the penicillin will exert a bactericidal or a bacteriostatic effect on the organism under consideration. This also is dependent upon the sensitivity of the particular organism under consideration. It is thus possible by varying the blood-tissue concentrations of penicillin to alter its activity in such a way as to "rèduce the rate of growth," "slightly kill the organisms," or "kill the organisms at a maximum rate."

■ Treatment of Tic Symptoms

Q.—I have a patient with typical tic symptoms with a trigger zone in the lower lip on the left side. The patient absolutely refuses to permit the neuro-surgeon to section the sensory root of the fifth nerve at the gasserian ganglion. What local treatment might be effective?

A.—As a diagnostic procedure a mental injection should be given by pressing a needle into the mental foramen and depositing local anesthetic solution. If the patient is entirely relieved of the pain by this procedure it might be assumed that control of the nerve at this point would give relief. An injection of alcohol solution in this area will give control of the difficulty for a period of 12 to 18 months if injected directly into the nerve. The best solution for nerve injection is that described by Blair and Ivy in *Essentials of Oral Surgery*: procaine, 2 per cent; alcohol, 70 per cent; chloroform, 5 per cent; water 23 per cent. This solution is effective and offers the advantage of being anesthetic in its action which prevents a great deal of the severe pain incident to an injection of this type.

Another very effective method of treating a case of this type is to make an incision in the labial sulcus in the region of the first and second bicuspid, through which the mental nerve is exposed. The mental foramen is identified and the nerve is grasped by a hemostat and is evulsed as far as possible. The nerve is then injected with 95 per cent alcohol and is cut at the mental foramen. As large a section as possible is cut from the course of the nerve. In many cases this gives permanent anesthesia and seems to be much more effective in its action than the injection technic alone. This technic is also applicable to the tic involving the infraorbital branches of the second division of the fifth nerve.

■ Clinical Value of Vitamin B_{12}

Q.—What is the clinical value of vitamin B_{12}?

A.—The work of West, who recorded his experiences with three cases of Addisonian pernicious anemia (Science, 107:398, 1948), states that vitamin B_{12} produces a positive hematological response in cases of Addison's pernicious anemia. Spies and others (South. M. J., 41:522, 1948), state that the patients experience a "great clinical improvement." These authors report that one case of nontropical sprue and two cases of macrocytic anemia exhibited positive hemopoietic activity as well as very marked clinical improvement. These patients noted "increase in their feeling of well being, mental alertness, strength and vigor, and complete relief of the soreness and burning of the mouth and tongue."

■ Absorbable Hemostatic Agents in Oral Surgery

Comment.—In the July, 1949, issue of the JOURNAL, under "Queries and Comments," a question was asked regarding the use of absorbable hemostatic agents in oral surgery. Part of the answer given was: "These products are a valuable aid in controlling hemorrhage, but as yet their value in the treatment of cysts and other large cavities has been disappointing and unproved." It seems to us that your statement may be misleading and in contradiction to the results which we, and others, have reported. In our original reports[1-3] we demonstrated the effectiveness of absorbable gelatin sponge in large cavities. Time and further experience have confirmed our original enthusiasm, rather than disappointed us. We place Gelfoam in every cystic cavity for the twofold reason of obliterating dead space and controlling bleeding. The results have been extremely gratifying for us and the patients. Postoperative treatment, other than removal of sutures, has been virtually eliminated. We consider this to be an important advance in oral surgery, with the prolonged period of changing dressings being no longer necessary. Silverman's recent paper[4] would seem to lend substantial support to the thesis that gelatin sponges and presumably Fibrin Foam are well tolerated by oral tissues, and facilitate healing.

1. Guralnick, W. C., Absorbable Gelatin Sponge and Thrombin in Oral Surgery. *Am. J. Orthodontics* 32:792 (Dec.) 1946.

2. Guralnick, W. C., and Berg, Leo, Gelfoam in Oral Surgery. *Oral Surg., Oral Med. & Oral Path.* 1:632 (July) 1948.

3. Thoma, K. H., and Sleeper, E. L., Gelatin Sponge in the Obliteration of Cavities Resulting From the Excision of Cysts and Tumors of the Jaws. *Oral Surg., Oral Med. & Oral Path.* 1:24 (Jan.) 1948.

4. Silverman, L. M., An Investigation of Gelatin Sponge With Thrombin and Penicillin in the Treatment of Oral Surgical Wounds. *Oral Surg., Oral Med. & Oral Path.* 2:260 (Feb.) 1949.

We would be very much interested in learning why you are dubious about the use of absorbable agents in cystic cavities.—*W. C. Guralnick and E. L. Sleeper.*

Reply.—It is unfortunate that the answer given by the member of the Editorial Board in the above-mentioned instance was misinterpreted. It is believed that Fibrin Foam or Gelfoam are well tolerated by oral tissues, and that these substances are helpful in the management of certain problems. In discussing this subject with many of the leading oral surgeons in this country from time to time, it has been the consensus of opinion that the use of these substances has been disappointing in most cases, and that their use in obliteration of large surgical defects has been overrated. The material has very definite value in some cases as has been pointed out by the numerous able investigators mentioned.—*Editor.*

■ General Anesthetic

Q.—Which is the best general anesthetic for oral surgery when it is necessary to use cautery and diathermy?

A.—It is undesirable to use any of the explosive anesthetics in the presence of cautery or diathermy, even in the closed system of administration. It is possible there may be leaks which might lead to serious explosion. Probably the safest method of obtaining complete surgical anesthesia is by the use of thiopental sodium, introduced intravenously with the use of tracheal intubation in order to assure a controlled airway.

Tribomoethanol, as a basal anesthetic supplemented with local anesthesia, also would be effective and would not offer any explosive hazards. This type of anesthetic, however, is not as easily controlled as the thiopental sodium intravenously and might offer objections in those individuals who have a history of previous toxicity to the use of procaine.

■ Dentigerous Cyst: Follicular Cyst

Q.—Is a dentigerous cyst the same as a follicular cyst?

A.—No, these are separate entities. The dentigerous cyst is formed in the part of the enamel organ which is to be found over the occlusal surface in which the enamel formation has been completed and where the epithelial cells are undergoing retrograde changes. These cysts are in association with teeth that have either completely or partially developed.

The follicular cyst is formed by the enamel organ before the cells have differentiated to the point of forming enamel. If the follicular cyst develops from a normal enamel organ a tooth in the normal dentition will be missing. If the cyst develops from a supernumerary sprout of the dental lamina or epithelial rests all of the normal teeth will be present with a cyst forming between two teeth or in the ramus.

■ EXCERPTA MEDICA. World wide abstract service. Baltimore: The Williams & Wilkins Company.

Excerpta Medica is the collective title for a series of fifteen monthly journals containing pertinent and reliable abstracts in English from every available medical journal in the world and covering the fields of clinical and experimental medicine. The sections deal with anatomy, physiology, endocrinology, microbiology, general pathology, internal medicine, pediatrics, neuropsychiatry, surgery, obstetrics and gynecology, otolaryngology, ophthalmology, dermatology, radiology and tuberculosis.

This monthly publication was subsidized by the government of the Netherlands in 1947. The monthly abstracting service covers articles from every available medical and oral surgery journal in the world. Numerous articles of interest in the field of oral surgery are to be found in Section IX, Surgery. This material is collected by hundreds of doctors throughout the world who cooperate in the project.

Dr. M. W. Woerdeman, Professor of Anatomy and Embryology at the University of Amsterdam, is the Chief Editor of this journal. *Excerpta Medica* was recently reorganized on a nonprofit basis and it is possible that additional support for further development of the various abstract services will be gained from U.N.E.S.C.O. in April.

Specialists in the field of medicine and surgery will find *Excerpta Medica* the most comprehensive and prompt abstracting of the world's current medical literature available.

This abstracting service has been approved by the American Medical Association and references will be made in the *Index Medicus* indicating abstracts that will be easily available in *Excerpta Medica*. This should greatly enhance both the value of the *Index Medicus* and *Excerpta Medica*. Numerous articles from the world's literature of interest in the field of oral surgery are abstracted in each monthly publication. These include articles on anesthesia and analgesia, surgical physiology and pathology, preoperative and postoperative therapy, surgical technic, surgical infections, antibiotics, physical medicine, fractures, heat and cold injuries, wounds and experimental medicine.

Section IX, Surgery, published in 1,920 pages per year at $25.00.

■ PAIN. Harold G. Wolff, M.D., and Stewart Wolf, M.D. 86 pages. Price $2. Springfield, Ill.: Charles C Thomas, 1948.

The origins, mechanisms of transmission and inhibition of painful sensations are discussed briefly, but with remarkable thoroughness. Sample chapter headings such as "Characteristics of Pain as a Sensation," "Pain Arising from Various Bodily Structures," "Control of Perception of and Reaction to Pain by Drugs" and "Surgical Measures for the Relief of Pain" indicate the scope of the book.

A large proportion of all pain is associated directly with the head, and much of it may be characterized by oral symptoms or pathology. This book is therefore pertinent to dentistry and should be interesting and useful to those dentists who will take the time to read it carefully.

■ ORAL ANATOMY. Harry Sicher. 529 pages with 310 illustrations including 24 in color. Price $15. St. Louis: C. V. Mosby Company, 1949.

The author emphasizes the serious problem in undergraduate dental education of correlating the subject of anatomy and other basic sciences to the clinical practice of dentistry in the subsequent clinical years. In *Oral Anatomy* an attempt is made to bridge the gap between theory and practice and prove that anatomic understanding facilitates clinical work and permits the substitution of adaptable and progressive

action for a rigid clinical technic. The text is designed to supplement rather than replace textbooks on dental anatomy and on human anatomy which are lacking in applied oral anatomy.

The book is divided into two parts comprising approximately 362 pages of descriptive anatomy and 108 pages of regional and applied anatomy.

Chapter 1 devotes 112 pages to the anatomy of the skull and in addition includes a functional analysis of the facial skeleton, extensive remarks on anthropology, cephalometry and craniometry which are of interest and value to dentists. The growth of the skull and of the facial skeleton is described and discussed in detail and constitutes most interesting and valuable information for the dentist and oral surgeon. In this chapter are also included sections on facial growth and tooth eruption and the growth of the pneumatic cavities of the skull. Numerous practical considerations are included in this chapter.

Chapter 2 is concerned with the muscles of the head and neck including the muscles of mastication and the superficial muscles of the head and neck, mouth and nose. Much important information for the oral surgeon is presented in this section.

Chapter 3 on the anatomy and function of the temporomandibular articulation is replete with sound information and analysis and is worthy of repeated study by dentists in all specialties of dental practice.

Chapter 4 covers the viscera of the head and neck. In this section is included an extensive presentation of the anatomy of the teeth, both permanent and deciduous, as well as the eruption of teeth, growth of jaws and occlusal considerations of particular value to the dentist. The latter portion of this chapter covers the anatomy of the soft palate and pharynx including the lymphatic tissue and the anatomy of the nose, paranasal sinuses and larynx.

The blood vessels of the head and neck are described and adequately illustrated in Chapter 5.

Chapter 6 is concerned with the lymphatic system of the head and neck and is most informative.

The nerves of the head and neck are described in Chapter 7 in easy reading style with fine illustrations to accompany the text.

Throughout the seven chapters of Part I the author has included many practical remarks which maintain the interest of the dental student and the dentist in an otherwise difficult subject.

Part II is devoted to regional and applied anatomy. Chapter 8 is a unique and unusually valuable description of the palpability of the facial skeleton.

In Chapter 9 the structure and relations of the alveolar process are of practical value to the oral surgeon. The illustrations are excellent.

Likewise, Chapter 10 on the anatomy of local anesthesia will be read with great profit by the oral surgeon.

Chapter 11 on arterial hemorrhages and ligation of arteries opens with a section on danger zones in the oral cavity, and the approach and technic of ligation of arteries are described in detail.

Chapter 12, the propagation of dental infection, is an excellent discussion of the pathways of infection and the fascial planes and spaces of the head and neck. Thrombophlebitis of the face and cavernous sinus thrombosis are included in this chapter.

Chapter 13 is an excellent discussion of tracheotomy and laryngotomy.

Chapter 14 is devoted to the temporomandibular articulation, covering overclosure and distal displacement, dislocation and the surgical exposure of the joint.

The final chapter presents the problems of atrophy of the jaws of the edentulous mouth.

This text can be highly recommended for dental students and practitioners. It will be found of great practical value to oral surgeons. —*C. W. Waldron.*

■ Medical Etymology. O. H. Perry Pepper. 263 pages. Price $5.50. Philadelphia and London: W. B. Saunders Company, 1949.

The author calls attention to the new and bewildering terminology that confronts the students of dentistry and medicine throughout

their years of study. The purpose of this handbook is to lessen the difficulties of medical terminology and to help to interest students in the words employed and in their proper way.

Part I, the introduction, presents the background of medical terminology in an interesting historical manner. Included is a detailed discussion of prefixes, suffixes, compound words and transliteration that is most informative. Consideration of eponyms and onomatopoetic words concludes the Introduction, study of which is necessary to appreciate Part II, Preclinical Subjects, Part III, Clinical Subjects, and Part IV, Dentistry.

Part II, Preclinical Subjects, covers the terminology of anatomy, including embryology, histology and neuroanatomy, physiological chemistry, physiology, pathology, pharmacology and therapeutics, and parasitology. The reader will find much of interest and value in these sections and will be intrigued by the complex derivation of a large number of terms in common usage.

Part III explains the terminology of the clinical subjects of medicine, surgery, pediatrics, neurology, gynecology and obstetrics, ophthalmology, dermatology, otolaryngology, psychiatry and radiology in a most informative manner. For the most part one cannot help being impressed by the fundamentally sound and descriptive combinations that make up medical terms. A number of terms might be considered far-fetched, illogical or even misleading in their derivation, but by common usage they have assumed definite meanings. Many terms have interesting and colorful backgrounds.

Part IV covers dental terminology. The author comments on the Greek and Latin words from which a large number of terms have been compounded resulting in the use of two series of synonyms—one Greek, the other Latin. Thus the student is faced with two names for many conditions. The term stomatology is favored as a more inclusive term in substitution for the term dentistry. Dental students will find this chapter of great assistance in understanding the many terms used in dental practice and literature. The derivations are interesting and informative even though in the opinion of the author many of these words have little historical interest. Terms used by the dental student in anatomy, pathology and other basic sciences covered in Part II and of clinical subjects included in the dental curriculum found in Part III are not repeated in Part IV, Dentistry.

This book is not a dictionary, but its less than 4,000 terms do represent almost all of the roots the student will encounter. One may question the author's implied concept that actually a student may not need a medical dictionary. Modern medical dictionaries contain so much useful, readily found information, in addition to the definition of words, that one should be part of the professional library of every student and practitioner of dentistry or medicine.

The author includes suggestions for use of this text on medical etymology. If followed the student will find the constantly occurring parade of new words of much greater interest when their derivation is understood.—*C. W. Waldron.*

■ ORAL BACTERIAL INFECTION. Lyon P. Strean. 185 pages, 57 illustrations. Price $5.50. Brooklyn, N. Y.: Dental Items of Interest Publishing Co., Inc., 1949.

According to the preface it is the author's intention to supply a ready reference book for practitioners requiring certain basic information quickly and for medical and dental students preparing for final examinations. In addition he proposes to deal with some of his own investigations concerning lactobacilli and the Hemophilus pertussis in some detail, and he also proposes to provide research workers and others with information about the methods employed in the study of bacterial endotoxins, exotoxins and toxoids. The subtitle promises that this text will be useful in diagnosis and treatment.

Both the content and the carefully prepared index supply adequate support for the first objective. This information is supplied in brief outline form frequently illustrated by outline, black and white drawings. The making of culture media, the preparation of stains and the technical cultivation of bacteria on artificial

media are described in considerable detail. There is a separate chapter on antibiotics and another on immunity and hypersensitivity. The author's description of his own investigations is brief and wholly without references to the original publications. The text on toxins, endotoxins and toxoids is generous from the standpoint of students and general practitioners but insufficient for use by technicians and research workers. The book is not a very helpful guide in diagnosis and treatment. Of the 57 illustrations, 16 are reproductions of photographs of not more than mediocre quality which add very little to the text.—*Edward H. Hatton.*

■ Plastic Surgery of the Nose. A. P. Seltzer. 305 pages, 221 illustrations, 7 color plates. Price $12. Philadelphia: J. B. Lippincott Company, 1949.

This easily read book is well arranged and contains ample illustrations of fine quality to accompany the subject matter of the text. A bibliography is found at the end of each chapter and the index is complete.

The first three chapters are devoted to the history of plastic surgery, the nose and the fine arts, and the nose in physical anthropology respectively. They contain much of interest and value to the plastic surgeon.

Chapter 4 presents a well illustrated, concise and informative description of the applied anatomy of the external and internal nose.

The embryology of the nose is adequately covered in Chapter 5.

The physiology of the nose, Chapter 6, is a well organized discussion of the respiratory airway in the normal and in the deformed nose, with accompanying illustrations. In addition, the functions of moistening, warming and cleansing the inspired air are covered in detail.

Chapter 7, preliminary and postoperative measures, includes an informative discussion of the psychologic factors and types of patients. Preoperative physical examination and complete records are stressed. The preparation of the patient, the field of operation, anesthesia, operating room technic and postoperative routine are discussed in great detail.

Fully illustrated instructions for making a facial mask are presented in Chapter 8.

Chapter 9 contains a description of the instruments favored by the author.

The various considerations pertaining to grafts of bone or cartilage in plastic surgery of the nose are found in Chapter 10. The technics for securing material for grafts are presented in detail. Included in this chapter is an ample discussion of skin grafts for surface defects of the nose.

Chapter 11 contains a lengthy detailed description of all aspects of a typical rhinoplasty. Operative procedures are profusely illustrated and described step by step. Semitransparency type drawings of the steps of operation are most effective. A detailed description of the immediate postoperative supportive dressings and the postoperative management are included in this chapter.

Chapter 12 presents a similar well illustrated description of the conventional submucous resection of the nasal septum.

Chapter 13 is devoted to the treatment of saddle nose of the more simple types, and a short discussion is included on measures necessary in cases with considerable scar tissue and deficiencies of skin.

Chapter 14 covers many nasal deformities and malformations of hereditary, developmental and acquired types including those due to disease and tumors. Included in this chapter is a section on the plastic repair of the deviated septum associated with a deflected tip of the nose.

The restoration of the subtotal and total loss of the external nose by surgery or prosthesis is discussed in Chapter 15.

Chapter 16 is devoted to surgery about the nares, covering the abnormalities of the columella, alae and nasal floor with numerous illustrations depicting the surgical procedures. The treatment of atresia and partial or total alar loss is included. Cleft lip and associated nasal deformity is discussed briefly in Chapter 17. Operations for repair are described.

The two final chapters, 18 and 19, cover postoperative complications of rhinoplastic surgery and a discussion of the unsuccessful operation, its causes and prevention. Numerous prac-

tical points are included in the text to assure better results and reduce the number of revisions required to secure a satisfactory appearance.

This worthwhile text is a valuable addition to the literature of this subject and is recommended to maxillofacial surgeons who are interested in the plastic surgery of the nose.—*C. W. Waldron.*

■ DETAILED ATLAS OF THE HEAD AND NECK. Raymond C. Truex and Carl E. Kellner. 160 pages. 147 plates, 116 in color. Price $15. New York: Oxford University Press, 1948.

This comprehensive Atlas provides pictorial guidance to all surgical specialists of the head and neck. The key to its practical use lies in its serial arrangement of regional dissections (many of which are approximately life size), progressing from more superficial to more deeply placed layers. Surface landmarks are emphasized and successively the deeper anatomic layers and structures are shown to clarify the interrelationships so important to the oral and maxillofacial surgeon. The drawings were made from original regional dissections using the same specimen for showing the anatomy of each series. The figure titles designate the series of dissection and the figure numbers show the progression to the deeper layers. The degree of enlargement or reduction from normal size is given, and each series of dissection is shown in the same size. This arrangement affords an accurate visualization of the laminar arrangements of fascia, muscles, bones, nerves and vessels of the head and neck. Study of these structures combined with that of the frontal and transverse sections enables the surgeon to acquire a practical three dimensional appreciation of orientation and depth. Cross references with the figure legend direct the reader to other views of each important region.

Of particular interest and service to oral surgeons are Figures 6 to 12, pages 7 to 13, of the head and neck; 17 to 25, pages 18 to 26, the face, infratemporal fossa, floor of the mouth, lateral pharyngeal space and pterygopalatine region. Figures 52 to 68, pages 50 to 63; 75 to 82 C, pages 67 to 72, include numerous dissections of interest to oral surgeons although primarily of particular importance to other specialists.

Figures 88 to 104, pages 76 to 90, show the detailed osteology of the head and jaw. The remaining divisions of the Atlas are devoted to frontal sections, Figures 105 to 116 A, pages 92 to 113, and transverse sections, Figures 117 to 136, pages 116 to 135. Many of these illustrations will be found of great service in enabling the surgeon to appreciate depth and relationships of important structures at the various frontal and transverse levels and will serve as a guide in the surgical approach to tumors or infection.

Anglicized terms have been used (Birmingham revision) as they are more anatomically correct and are more descriptive. The term submandibular is used instead of submaxillary in identifying the gland, and maxillary artery and facial artery replace the respective terms internal and external maxillary arteries. Twenty-six pages of index enhance the practical value of this exceptionally well illustrated Atlas.

The increasing accumulation of pathological, physiological and clinical knowledge of regions of the body such as the head and neck confirms the opinion that anatomy is no longer a catalog of names but enters into the understanding and interpretation of symptoms and when mastered renders surgical interference both effective and safe.

This Atlas is recommended for dental and medical students and practitioners and for surgeons interested in this field. Oral and maxillofacial surgeons will find it most useful.—*C. W. Waldron.*

■ The Bone Bank (editorial). Paul R. Lipscomb. Surg., Gynec. & Obst. 89:485, October, 1949.

The author states that a department of spare parts seems destined eventually to be a part and parcel of every modern hospital, just as such a department is today an integral part of every modern garage. It seems that in most instances human tissues are best preserved for future use by means of refrigeration. Some workers feel that it is not necessary to preserve the organic element of bone in a viable state. Some surgeons use homogenous bone transplants preserved in weak aqueous solution of Merthiolate in a manner similar to that used for a number of years to preserve transplants of cartilage. A solution of 1:1,000 aqueous Merthiolate has been used to maintain sterility and likewise homogenous bone has been preserved in alcohol and other antiseptic solutions. Fresh or dry bone sterilized by boiling or autoclaving has also been used. Bone preserved in this state can only serve as a source of inorganic calcium and phosphorus and as a trellis or framework for the process of "creeping substitution." The author quotes Dr. A. C. Walsh, "In preserving tissues for transplantation at a later date, three principles must be observed:

"1. The cells of the tissues should be kept viable.

"2. If viability is not possible or necessary, the tissue should be preserved from degenerative changes.

"3. The tissue must be maintained in the same aseptic state as it was when placed in the preserving containers.

"From a study of the literature it would appear that all these principles can best be fulfilled by preservation at a temperature below −20° C. Above this degenerative changes occur and sterility cannot be assured. Temperatures below −25° C. appear to be of no practical value."

The author states that probably none of the bone cells of refrigerated bone transplants remain viable after being transplanted into the host. It is also felt that most of the cells of the autogenous fresh bone undergo degenerative changes. It is felt that any bone graft has its value in the support and fixation which it offers and the framework on which new bone is to develop using in part the mineral salts in the bone transplant. The author feels that the same result is obtained whether autogenous or frozen homogenous transplants are used. It is agreed by most workers that the autogenous transplants result in a satisfactory end result sooner than the homogenous transplants. Most of the bone placed in the bone bank is in the form of segments of rib removed during the course of combined thoracolumbar sympathectomy and other thoracic operations. Traumatic amputations and fresh cadavers also may be a source of unlimited supplies of bone for use in the bone bank. The bone is removed under aseptic conditions and is immediately placed in a sterile container with a sealed cap. This container is placed in a second sterile container or wrapped in sterile surgical linen. It is then immediately placed in a deep freeze unit which maintains a constant temperature of −20 C. to −25 C. The bone is left in the freezer until a few minutes before the time when it is to be used. Walsh believes that the bone will remain sterile and viable for an indefinite period, although the author hesitates to use bone more than three months old.

The blood group of the donor does not need to be compatible with that of the recipient as far as the use of homogenous bone transplants is concerned. The donor must be free from a history of syphilis, and serologic tests must rule out this disease, for it has been shown that the *Treponema pallidum* may survive at very low temperature. The donor should not have jaundice, infectious or malignant neoplastic illness. The age, sex, nationality, occupation, past and present illnesses of the donor should be filed for future reference.

Homogenous bone grafts from a bank spare the patient shock, increased morbidity and

cost of a second operative procedure. This is especially useful in children and is used in filling large cystic defects, defects from giant cell tumors and for reinforcement of autogenous grafts. The author believes that the autogenous and homogenous transplants used together probably give the most effective end results. The author believes that fresh autogenous bone is the material of choice in any bone grafting procedure but in certain cases where it is difficult or inconvenient to remove bone from the patient that the frozen bone from a bank would be quite satisfactory. He believes that we are not justified at this time in using preserved refrigerated bone indiscriminately as a large enough series for adequate study is not yet available.

■ THE ANESTHETIC MANAGEMENT OF PATIENTS WITH FRACTURE OF THE MANDIBLE. S. G. Hershey and E. A. Rovenstine. Anesthesiology 9:381, July, 1948.

The anesthetic management of patients with fracture of the mandible, as is true with other anesthetic procedures, may vary with the person administering the anesthetic. There are, however, fundamental principles which must be observed if the most satisfactory results are to be obtained. Principles relating to preoperative preparation, selection of anesthetic agents, the working conditions of the surgeon and the complications both during and following surgical procedures, are principles with which the anesthetist must be familiar.

A workable anesthetic technic for surgical procedures about the jaw was developed over a ten year period. Three hunderd and sixty-one oral operations were used as a basis for the development of this technic, all being one of these types: (1) circumferential wiring of the fractured mandible over an acrylic splint (204 cases); (2) Roger-Anderson external fixation of the fractured mandible (122 cases), and (3) resection of the mandible for prognathism, with fixation to the upper jaw (35 cases).

Premedication consists of an average dose of morphine sulfate and scopolamine hydrobromide 90 minutes before operation and occasionally 0.1 Gm. of pentobarbital 45 minutes later.

A 4 per cent solution of cocaine is sprayed into each nostril ten minutes preoperatively and again in five minutes. The total amount of cocaine sprayed into the nostrils is less than 5 cc. This facilitates the introduction of a soft rubber endotracheal catheter through which a small amount of cocaine is then sprayed to anesthetize the glottis and trachea. Blind nasotracheal intubation is then accomplished without undue discomfort to the patient.

Following intubation, a 2 per cent solution of thiopental sodium is given intravenously until the patient is unconscious. The use of thiopental sodium serves three purposes: (1) it produces rapid, quiet induction without causing pain usually experienced in jaw injuries when the face piece is used; (2) it protects the patient from any delayed cocaine reaction, and (3) it decreases the probability of severe emergence delirium.

After induction of anesthesia with thiopental sodium, the throat is packed with moist gauze and cyclopropane anesthesia is begun. If there are any contraindications to the use of cyclopropane, ether is the anesthetic of choice.

This technic was not followed in every detail on all the cases. Some patients were not intubated while awake. In these cases it was not necessary to use cocaine and thiopental sodium. The anesthesia was induced with cyclopropane and then intubation was performed. In case of injury to the nose, oral rather than nasal intubation was used. Occasionally block anesthesia was used, but this method proved to be less desirable from the standpoint of the patient's comfort.

The study revealed that two complications warranted special considerations: (1) emergence delirium which, if not controlled, may undo all that has been accomplished during the operative procedure, and (2) aspiration and suffocation resulting from postoperative vomiting. The first, common in alcoholic patients, may be controlled by premedicating the patient with intravenous apomorphine (1.5 to 2.0 mg. in 10 cc. of saline solution). The second complication, especially applicable to patients requiring intermaxillary wires, may be avoided

by using intermaxillary rubber ligatures to immobilize the mandible to the maxilla until the patient is completely conscious. A nurse or intern should be placed in charge of the unconscious patient in order that the rubber ligatures may be removed quickly should circumstances warrent.—*De Orr Cooley.*

■ Carcinoma of the Lip. J. E. Cross, E. Guralnick and E. M. Daland. Surg., Gynec. & Obst. 87:153, August, 1948.

The authors present an interesting statistical review of 563 case records of carcinoma of the lip at the Pondville Hospital from June, 1927, to December, 1941. These cases represent 2.9 per cent of the 19,664 new patients examined in the outpatient department during the period covered by the study. It was hoped that by a critical study certain criteria could be established from which it would be possible to outline the method of therapy most likely to be successful in a given case and to give a reasonably accurate prognosis at the outset of treatment.

It was found that this pathologic condition occurred in the ages 25 to 91, the median age being 62. Ninety-eight per cent were men and 2 per cent were women. Seven and two tenths per cent had a positive serology test.

Of the total group 497 occurred in the lower lip, 19 in the upper lip, and 47 in the labial commissures. The median duration of their cases prior to consultation was six months. Seventy-six and a half per cent had lesions with a maximum diameter of 2 cm. or less, 39.2 per cent measured 1 cm. or less.

Ninety-four per cent of the cases were habitual tobacco users, with 71 per cent of these smoking a pipe. Fifty-eight per cent had poor oral hygiene, 14.5 per cent had leukoplakia or keratosis, 11.9 per cent had other unassociated carcinomas. Sixty-two patients gave a history of previous trauma.

This hospital favored surgical therapy, doing the excision of the lip lesion and dissection of the cervical lymph nodes at one operation. They now prefer to bring the primary lesion under control before attacking the lymph nodes. It is felt that since lymph node metastases from carcinoma of the lip occur by way of emboli rather than by lymphatic permeation, *en bloc* dissections of the primary lesion and the regional lymph node bearing areas are unnecessary. This enables a better evaluation of the status of lymph nodes by allowing inflammatory nodes to become quiescent after the eradication of sepsis within the mouth and incident to the tumor itself. The supraomohyoid technic is done when neck dissection is carried out, or in cases with more extensive metastases a more radical dissection down to the level of the clavicle is done.

Their statistics for the total unselected cases revealed three year cures in 67.1 per cent and five year cures in 63.6 per cent. Of the patients who received all their treatment at Pondville Hospital 93 per cent achieved three year cure status through surgical treatment, 47.5 per cent through radiation, 58.3 per cent through combined treatment; giving a total of 81.4 per cent. Of this latter group five year cures were slightly less. They feel that three year cures are as significant statistically as five year cures in carcinoma of the lip.

Their statistical evidence lends support to the fact that carcinoma of the labial commissures are approximately twice as difficult to control as lesions in other locations on the lips.

The figures derived also show that curability of carcinoma of the lip is directly related to the size and pathologic grade of the primary tumor and the presence (or absence) of lymph node metastases.

A three year cure stage was attained in 35.9 per cent of the patients who underwent neck dissection with pathologically proved cervical lymph node metastases.—*A. S. Lasky.*

■ Recent Advances in Antibiotic and Sulfonamide Therapy. Harrison F. Flippin and William P. Boger. Virginia M. Monthly. 76:56, February, 1949.

According to the specific organism under treatment, the penicillin plasma concentration produces three phases affecting bacteria. When a massive dose is administered, there is first a

bactericidal phase. As the concentration falls a bacteriostatic phase is produced. The third phase is a suppressive or metabolic concentration during which the bacteria may be damaged by the interference of penicillin with the enzyme systems of the organisms. Although they are able to multiply the rate of multiplication is less than normal. When massive doses are given a plasma concentration greater than the required bactericidal concentration is produced resulting in a waste of the drug, and a zone phenomenon may result if this concentration is exceeded. This is an experimental concept.

Some bacteria are killed by penicillin only while they are multiplying, hence bacteria in a physiologically inactive stage are affected only when multiplication is resumed. A constant bacteriostatic concentration results in less antibody formation than if treatment is intermittent, in which case opportunity is afforded for some multiplication of the bacteria. It is doubtful in any event that bactericidal concentration will kill all the organisms. The body defense actions must not be overlooked in the control of these infections.

The duration of these three phases depends on the sensitivity of the organism. If the death-growth cycle is short, a bactericidal concentration must be maintained more frequently, and the reverse is true. Optimal penicillin therapy would consist of an initial dose large enough to produce a maximal bactericidal concentration and subsequent doses at intervals to kill the remaining organisms during a phase of multiplication.

Streptomycin and the sulfonamides may be fitted into this picture. Streptomycin produces a decided bactericidal action with the disadvantage that subsequent doses produce resistance of the organism. The synergistic action of both streptomycin and penicillin in an initial dose gives a greater bactericidal effect than either of the drugs singly. Sulfonamides, being primarily bacteriostatic, can be used to maintain bacteriostasis so that subsequent doses of bacteriostatics will reduce further the number of surviving organisms.

Plasma concentrations of crystalline penicillin in aqueous solution (300,000 units) can be demonstrated for eight to twelve hours, comparable doses of procaine penicillin in aqueous solution for twenty-four to forty-eight hours. "Caronamide," a drug which inhibits the renal excretion of penicillin, given 2 to 3 Gm. every three to four hours will elevate the plasma concentration from 2 to 7 times.

Sulfonamide combinations increase their solubility in urine, reducing the hazards of crystals or calculi, thereby eliminating the need for adjuvants.

A combination of sulfonamides (sulfadiazine, sulfamerazine) has proved to be equal to sulfadiazine alone, with regard to therapeutic results achieved, and superior to sulfadiazine with regard to urinary toxicity. Triple sulfonamide combinations have indicated that the occurrence of crystalluria can be decreased to negligible proportions, and may eventually replace the double combinations.—*W. H. Justi.*

■ CYSTS OF THE NASAL VESTIBULE. Fernand Montreuil. Ann. Otol., Rhin. & Laryng. 58: 212, March, 1949.

Cysts of the nasal vestibule are generally considered to be of rare occurrence. There is at present no conclusive evidence that they show any predilection as to sex or race. They appear as frequently on the right side as on the left side. These cysts may be found during a routine examination of the nose, but they are more commonly brought to the attention of the doctor by the patient who may complain of a unilateral nasal obstruction, a lateral nasal swelling, or a feeling of fullness of the upper lip. It is not common to find pain associated with this form of cyst unless there is secondary infection present.

Examination usually reveals a unilateral protrusion of the upper lip and a flattening of the nasojugal fold. An obstructive mass on the floor of the nose just posterior to the nasal vestibule may be observed. The size of this mass may vary from one that is scarcely visible to one that causes complete obstruction. Intraoral examination often reveals a fullness in the vestibule on the affected side. Palpation reveals a smooth, nontender, partially mobile

and slightly depressible mass. It is often partially fixed to the nasal mucosa. The aspirated content of the cyst is a sterile mucoid straw-colored fluid, there being no cholesterol present.

Those pathologic conditions which should be considered in a differential diagnosis are: (1) furuncle of the nasal vestibule; (2) cysts of dental origin; (3) Gerber's pad; (4) cysts of nondental origin.

Cysts of the nasal vestibule may be differentiated from a furuncle on the basis of a history of the existence of a lump for several months previous to the date of examination.

These cysts may be differentiated from cysts of dental origin on the basis of a thorough examination (including roentgenologic examination) of the teeth and supporting bony structures. In the case of a dental cyst, roentgenograms will show it to be associated with either the root or crown of a tooth. In the case of a radicular cyst, the tooth will be nonvital.

Roentgenologic examination of the maxillary anterior teeth will be adequate in the diagnosis of Gerber's pad.

Cysts of nondental origin to be included in a differential diagnosis are globulomaxillary cysts and incisive canal cysts. Roentgenologic examination will usually suffice in differentiating between these two types of cysts. The first will be located between the roots of the lateral and cuspid teeth and usually causes a divergence of their roots. The second is located in the midline between the roots of the maxillary central incisors. This cyst usually projects intraorally, but may extend nasally, in which case it will be in close proximity to the septum rather than the lateral floor of the nose as in the case of a cyst of the nasal vestibule.

It is now believed that cysts of the nasal vestibule have an embryologic origin. Bruegger-mann presents the theory that they arise from an anteriorly misplaced lacrimal duct. Klustadt believes that these cysts have two origins: (1) from the facial cleft, and (2) from the lacrimal duct.

The treatment of choice is complete surgical excision. The ideal site of approach is through an intraoral incision in the gingival mucosa medial to the Caldwell-Luc incision.—*De Orr Cooley.*

■ A Study of Novocain (Procaine) Sensitivity Among Navy Dental Officers. W. V. Charter, C. A. Schlack, and R. L. Ware. J. D. Res. 28:337, August, 1949.

A study has been made by the United States Navy Dental Corps in an effort to determine procaine sensitivity among Navy dental officers. The purpose of this study was to obtain data for dental logistics in the Navy, but it also brings out information that is of value to the oral surgeon, the general dental practitioner, the dermatologist, and the pharmacologist.

Questionnaires were sent to approximately 7,500 dentists representing the Naval Dental Corps which included those in the regular Navy, the reserve officers on active duty, and the reserve officers on inactive duty. Fifty-two per cent of the questionnaires were returned and are the basis of this report.

It was found that a sensitivity rate of 3 per cent was constant for each of the groups listed. In relation to age the older dentists had a greater prevalence of sensitivity than the younger representatives of the report. Eighty-two per cent of the sensitive group reported only a dermatitis, 7.8 per cent of the sensitive group reported urticaria and the remainder of the sensitive individuals reported other types of manifestations. The sensitive group was composed of 128 dentists. Of this number only four individuals reported that they were prevented from performing all operating procedures, and 49 reported symptoms regularly upon exposure. Forty-three per cent of the sensitive cases lasted less than one week, while 1.1 per cent of the cases lasted continuously while being exposed to the drug. Those having regular attacks upon exposure tended to maintain symptoms for longer periods than those reporting only occasional attacks. There was a significant difference in occurrence of attacks between the officers who prepared solutions from tablets or crystals and those who routinely used commercially prepared cartridges. The only entirely effective countermeasure that was tried was the discontinuance of the use of Novocain.

The continued use of the word Novocain in this article makes one wonder whether the authors were specifying a particular brand of

procaine, or whether through common usage procaine in general is meant. While the authors recognize many limitations in this investigation, it is a worthy report of what may be classified as one of the occupational diseases of dentistry. —*C. R. Petty.*

■ MANDIBULAR RECONSTRUCTION, WORLD WAR II. T. G. Blocker and Roy A. Stout. Plast. & Reconstruct. Surg. 4:153, March, 1949.

This article deals with the reconstruction of the mandible by use of bone grafts, describing the technic and follow-up results of a large series of cases treated in World War II. The author emphasized that the iliac crest, in addition to providing a large amount of readily available cancellous bone, was found to be particularly suitable for use in mandibular restoration because it may be readily shaped to conform to the normal contour of the jaw, and may be useful in providing a sufficient foundation for dental prosthesis.

A total of 1,010 mandibular bone grafts were reported: 836 were from the ilium; 151 were from the rib; 23 were from the tibia.

The best results were obtained when at least six months had elapsed from time of injury until time of the bone grafting procedure. Although many types of material were used for fixation of the grafts, it was the opinion that stainless steel wire was best because of its inert properties.

Local anesthesia was the choice mainly because of the hemostatic properties and fewer possibilities of postanesthetic complications.

The average time of immobilization was from six to eight weeks with an additional four weeks in some cases. The average statistics regarding failures was 11.2 per cent although the author admits that the figure would have been higher if the follow-up examination had been more complete. As usual, in any type of bone graft, the failures were accounted for by infection and failure of the surgeon to provide adequate soft tissue covering.

It was found later on during the war that simpler devices for fixation could be used; that is, multiple loop wiring and elastic traction, as effectively as complicated splints. Emphasis was laid on the use of pure cancellous bone as the choice of material for operative repair.

■ RESTRICTED JAW MOTION DUE TO OSTEOCHONDROMA OF THE CORONOID PROCESS. R. R. Shackelford, M.D., and W. H. Brown, M.D. J Bone & Joint Surg. 31A:107, January, 1949.

This is the authors' report of 4 cases of osteochondroma of the coronoid process.

The important points to emphasize in a diagnosis of osteochondroma of the coronoid process are: a slowly progressive, painless limitation of movement of the jaw; unilateral malocclusion of the teeth; a stony hard swelling in the region of the zygoma on the affected side; a bony projection, visualized by roentgenograms using a technic designed by the authors. The usual anteroposterior and lateral plates are not suited for a diagnosis of a lesion in this region.

In the 4 cases presented, only 1 presented a history of trauma to the jaw or face. The common history was of slow growth of the lesion on the affected side. Two of the patients had a "cracking" noise in their jaw on opening and closing; all displayed limitation of movement.

Treatment consisted of surgical excision of the lesion. The incision is made superior to, and parallel with the inferior border of the zygoma to avoid injury to the facial nerve. The zygoma is bared subperiosteally, and the desired portion is resected, allowing access for the removal of the lesion. In 2 of the reported cases, the resected portions of the zygoma were grafted into place; in the third and fourth cases, no attempt was made to restore the resected zygoma. The cosmetic results were equally good in all 4 cases. The patients regained full movement of the jaw. Postoperative examinations of these patients at intervals of from two to five years show restoration of normal function of the jaw, symmetry of the face and no sign of recurrence.

It is suggested that this lesion, although uncommon, occurs more frequently than is recognized.—*W. H. Justi.*

■ ANKYLOSIS OF THE TEMPOROMANDIBULAR JOINT. C. L. Straith and J. R. Lewis. Plast. & Reconstruct. Surg. 3:464, July, 1948.

The subject of ankylosis of the temporomandibular joint is discussed in a logical sequence as to the etiology, pathology, symptoms, diagnosis, treatment and after care.

The most common causes for the condition were thought to be trauma, direct or indirect, and infection in or about the joints. A comprehensive table of data on etiology, age of onset, duration of symptoms, degree of involvement, and so forth, was given for 16 cases treated at the Straith Clinic. The incidence as to sex seems to be slightly higher in men. According to Orlow 80 per cent of all cases are said to occur between 1 and 10 years of age, 15 per cent between 11 and 12 years, and 5 per cent in the 20 to 30 age group. In the authors' cases the age of onset in each occurred prior to the ninth birthday with an average of 2 years and 8 months.

The pathologic picture varied with the etiology and the site of involvement was made a basis for classification as follows: (1) the intra-articular type, (2) the extra-articular type, and (3) a combination of 1 and 2.

The most typical symptoms were inability to open the mouth and difficulty in eating or talking. The authors noted that the resultant mandibular deformity was in direct proportion to the age at which the condition occurred. When the condition occurred prior to the fifteenth year deformity can be expected. In unilateral ankylosis at an early age there is rounding of the cheek and a lateral backward displacement of the chin on the affected side. With bilateral involvement at an early age there is underdevelopment of the lower third of the face. These conditions predispose to poor intermaxillary relations, malocclusion, poor oral hygiene and malnutrition. Weight gains occurred soon after surgical treatment.

Diagnosis is arrived at by correlation of events of trauma or infection followed by limitation of movement of the mandible, evident signs and symptoms and good stereoscopic roentgenograms of the temporal joints in the lateral as well as posteroanterior views. They suggest that consideration of hysteria, tetanus, reflex spasms secondary to oral disease, acute parotitis, acute arthritis, meniscitis and mechanical restriction afforded by a fractured zygoma be included as possible differential diagnostic factors.

In the group of cases discussed the interval between time of onset and the visit to the surgeon averaged over seven years. The type of operations performed were osteotomy of the neck or ramus of the mandible and osteotomy of the coronoid process when involved. The bilateral cases were done in either one or two stages with the latter preferred. General or local anesthesia was used. An external approach through a hockey stick incision was preferred with the outline running from a point immediately anterior to the inferior attachment of the lobe of the ear, upward, anterior to the tragus and overlying the zygoma. One-half to three-fourths inch of bone was removed in each case. The condyle was removed when the osteotomy was made at the neck and when the part could be elevated out of the fossa. It was pointed out that manipulation of the mandible is advisable when osteotomy is complete in order to check for other unsuspected points of restriction of movement. Preserved fascia lata was used to fill the site of osteotomy and the wound closed with a rubber drain in place.

After the operation early active jaw motion was encouraged. On the second or third postoperative day the liquid diet was replaced by a regular diet and chewing gum suggested between meals. After about two weeks massage of the masseter muscles was started. Similar treatment was suggested for any areas affected by transient facial paralysis. After four to six weeks the patient was referred for necessary dental care and reconstruction. After such reconstruction was completed the patient was re-evaluated as to the need for further cosmetic correction. The most common residual deformity is that of an underdeveloped chin. The authors correct this defect by building up the site with flaked or diced cartilage, or with a properly contoured solid cartilage or iliac graft. The psychologic importance of the latter procedure is considered an essential part of the overall treatment in these cases.—*L. E. McKelvey.*

Deaths

HARRY MCFARLAND was born September 23, 1879, at Ness City, Kansas. He was graduated from the Western Dental College in Kansas City as valedictorian of his class in 1902. Dr. McFarland's interests were wide and varied. In addition to contributing to the field of oral surgery he took an active part in community affairs and was an ardent sportsman. He served as president of the American Society of Oral Surgeons in 1936 and recommended in his presidential address that the American Board of Oral Surgery be established.

He organized the Wyandotte County Dental Society of Kansas in 1913 and served as its first president. He was a past-president and honorary life member of the First District Dental Society of Kansas as well as of the Wyandotte County Dental Society. He was elected president of the Kansas State Dental Association in 1923 and during his term of office influenced the state legislature to create a law permitting boards of education to provide free annual dental inspections for public school children.

In 1927 Dr. McFarland who was already known nationally as a writer and lecturer was made a member of the faculty of the Western Dental College. He was an active and enthusiastic church worker, a Fellow of the American College of Dentists, and a member of Delta Sigma Delta and Omicron Kappa Upsilon dental fraternities. While president of the Kansas City Rotary Club in 1937 he represented his club at the International Convention of Rotary in Nice, France. He died in Kansas City, Kansas, June 28, 1949.

JASON L. LISCOM, one of Southern California's most distinguished dentists, died suddenly October 7, 1949.

Dr. Liscom was born at Rock Island, Illinois, April 27, 1901. He graduated from the University of Illinois in 1923 and immediately took up residence and practice in Los Angeles. Dr. Liscom was a life member of the Southern California State Dental Association; an Executive Councilor of that Association; a member of the Advisory Council of the Dental Medicine Seminar; a Diplomate of the American Board of Oral Surgeons; a member of Delta Sigma Delta fraternity; hospitality chairman of the Wilshire Kiwanis Club, and a Past Master of his Masonic Lodge.

Extremely active in all of his organizational affiliations, Dr. Liscom was very popular as a clinician and as a director of many ceremonial activities.

He leaves a void in Southern California dentistry and in American dentistry, for he had achieved national prominence both in the field of oral surgery and in fraternal work.

Announcements

■ Roy A. Stout Appointed to Staff of University of Texas

Colonel Roy A. Stout has retired from the Army Dental Corps after thirty-two years of service, and has been appointed associate professor of Surgery, University of Texas Medical School, Galveston, Texas.

Colonel Stout has long had an international reputation as an oral and maxillofacial surgeon. During World War II he was the maxillofacial surgical consultant in the European theater of operations. Since the war Colonel Stout has been Chief of Dental Service at army general hospitals designated as maxillofacial centers.

■ Appointments to the University of Southern California School of Dentistry

Charles M. Woodward, Pasadena, has been appointed professor of Oral Surgery at the University of Southern California School of Dentistry. Dr. Woodward has practiced as an oral surgeon since 1918. He conducts postgraduate courses in oral surgery through the Extension Division of the University of California.

Arthur E. Smith has been appointed professor of Reconstructive Plastic, Maxillofacial and Oral Surgery at the University of Southern California School of Dentistry.

H. Newman Brownson has been appointed professor of Oral Surgery and head of the Surgery Department at the University of Southern California.

■ Dental Internships Open at the Jewish Sanitarium and Hospital for Chronic Diseases

Dental internships are open at the Jewish Sanitarium and Hospital for Chronic Diseases. They are for one full year beginning July 1, 1950. They allow $50 per month with full maintenance. For further information write Leonard Kohn, D.D.S., Director, Dental Department, East 49th Street and Rutland Road, Brooklyn 3, N. Y.

■ New Grants for Dental Research Announced

Public Health Service grants totaling $22,897 for new dental research projects were made in October by the National Institute of Dental Research. These grants are in addition to grants announced in July, amounting to $160,708, for continuing dental research projects already under way. The list of new grants is as follows: University of Southern California, $9,843 for study of the growth and development of the face and the temporomandibular articulation under the stimulation of the adrenal cortex; University of Chicago, $3,456 for the study of oral lactobacilli associated with dental caries; Jewish Hospital of Brooklyn, $6,048 for the study of the composition of teeth and its relation to human dental caries; New York University, $513 for the study of the effects of standard drugs on dental tissues as indicated by P 32 measurements; Ohio State University, $1,575 for an experimental study of deformation and fracture patterns of the human mandible; University of Oregon, $1,462 for investigation of the effect of alloxan diabetes on the incidence of dental caries in the Syrian hamster and for clinical and laboratory examination of caries susceptibility in human diabetes before and after institution of treatment.

■ University of Washington Dedicates New Health Science Building

The University of Washington dedicated its new Health Science Building in Seattle, Sunday, October 9. The $9,000,000 building houses the University's new school of dentistry and medicine and its school of nursing that was established in 1917. Additions for the College of Pharmacy and a research hospital are planned for the immediate future. The first classes in dentistry and medicine, which will graduate next spring, have been held in other buildings while the new building was being erected.

Dr. Donald G. Anderson, secretary of the Council on Medical Education and Hospitals of the American Medical Association, gave the dedication address, Governor Arthur B. Langlie laid the cornerstone and Dr. Raymond B. Allen, president of the University presided.

One of the most spectacular rooms of the building is the U-shaped dental operatory which covers nearly a half acre of floor space and is occupied by 110 dental chairs and cabinets. The operatory, walled by sheets of glass and glass brick, is so large that when all the 220 volt dental lamps are shining a special cooling system is necessary to keep the temperature comfortable.

The new Health Science Building will be used by regular students and postgraduate and professional groups. Nearly 2,000 undergraduate and graduate students in dentistry, medicine and nursing will receive instruction in the new building this year in addition to 1,500 students from other university departments who will receive instruction in basic medical sciences.

Deans of the units in the Health Sciences Division are: Ernest M. Jones, dentistry; Edward L. Turner, medicine; Elizabeth Soule, nursing; Forest J. Goodrich, pharmacy.

■ Leo Winter Memorial Meeting

In memory of one of the foremost oral surgeons of our times, the Leo Winter Memorial Committee of the New York University Dental School Alumni Association has arranged a meeting on Wednesday, January 25, 1950, at 8:30 P.M. at the New York University Dental School, 209 East 23rd Street, New York City. Dr. Maurice J. Hickey, Associate Dean of the Faculty of Medicine, School of Dentistry, Columbia University, will be the essayist for the evening. He will speak on the present and future status of oral surgery. Dr. Harold O. Voorhis, vice-chancellor of New York University, will present the eulogy.

All members of the dental and allied professions are cordially invited to attend.

Professor Leo Winter was head of the Department of Oral Surgery and director of the Postgraduate Division of the College of Dentistry of New York University and chief of Dental Service of Bellevue Hospital. He died July 6, 1948.

■ Coney Island Hospital Dental Internship

Applications for a dental internship at the Coney Island Hospital may be had by applying to the superintendent, Coney Island Hospital, Ocean Parkway and Avenue Z, Brooklyn 23, New York. The internship is for one year beginning July 1, 1950. Examinations will be held during the Christmas Holidays.

■ Cameron Appointed to Staff of Graduate School of Medicine, University of Pennsylvania

James R. Cameron, head of the Department of Oral Surgery at Temple University and chairman of the Department of Oral Surgery at the Pennsylvania Hospital, has recently been appointed acting director of the course in Oral Surgery-Oral Anesthesia in the Graduate School of Medicine, University of Pennsylvania, Philadelphia.

■ Courses in Oral Surgery at the University of Michigan

The University of Michigan School of Graduate and Post-Graduate Dentistry announces the following courses in oral surgery:

Two week extension postgraduate course in minor oral surgery—January 16-27; February 13-24; March 21-April 7.

One year basic science training course in oral surgery in preparation for internship leading to qualification by the American Board of Oral Surgery—September 1-June 15.

Three year fellowship in oral surgery at the University of Michigan Hospital leading to Master of Science degree in oral surgery and designed to meet qualifications of the American Board of Oral Surgery.

These courses are available to graduates of recognized dental schools in good standing with the American Dental Association.

A two year fellowship in maxillofacial surgery is also announced. Applications will be received from men with both dental and medical degrees. For further information write Dr. Paul Jeserich, Director of the W. K. Kellogg Foundation, Institute of Graduate and Post-Graduate Dentistry, University of Michigan, Ann Arbor, Mich.

■ Residency in Oral Surgery at Louisville General Hospital

The Louisville General Hospital, Louisville, Kentucky, offers a residency in oral surgery as of January 1, 1950. The hospital has 450 beds and is affiliated with the University of Louisville Schools of Medicine and Dentistry. The staff consists of full time personnel, three interns and a resident. Training and instruction in anatomy, pathology and other basic sciences are designed to prepare candidates for the American Board of Oral Surgery. For further information write to Dean Raymond E. Myers, School of Dentistry, University of Louisville, 129 East Broadway, Louisville, Kentucky.

■ Postgraduate Courses in Dentistry

The Ohio State University College of Dentistry announces the following postgraduate courses in dentistry of oral surgical interest to be presented in 1950:

Oral Pathology	Hamilton B. G. Robinson	January 9-13
Anesthesia	M. Lowell Allison	January 16-20
Oral Surgery	D. P. Snyder	January 23-27
Anatomy	Linden F. Edwards	March 20-24

Each course is given for five consecutive days, Monday through Friday, and the fee for each is $50, except for Oral Surgery which is $100. All of the courses have been approved by the Veterans Administration. The classes are limited to ten men each, giving opportunity for discussion and instruction.

Further information and applications may be secured from the Postgraduate Division of the College of Dentistry, Ohio State University, Columbus, Ohio.

■ Annual Meeting of the Southeastern Society of Oral Surgeons

The first annual meeting of the Southeastern Society of Oral Surgeons will be held at the Biltmore Hotel, Atlanta, Georgia, March 17 and 18, 1950.

Editorial communications

■ ORIGINAL COMMUNICATIONS—This journal invites concise original contributions in the broad field of clinical and experimental surgery of the mouth and associated structures, including roentgenology, diagnosis, anesthesia and oral surgical pathology. Articles are accepted for publication with the understanding that they are contributed solely to the JOURNAL OF ORAL SURGERY. All manuscripts are subject to editorial modification. Neither the editors nor the publishers accept responsibility for the views and statements of authors expressed in their communications.

■ MANUSCRIPTS—Manuscripts submitted for publication should be sent to Reed O. Dingman, Editor, 221 North Ingalls Street, Ann Arbor, Michigan.

Manuscripts should be typewritten on one side of the paper only, with double spacing and liberal margins. The author's name and the abbreviated title of the article should appear at the top of each manuscript page. The original should be sent to the editor and a carbon copy retained by the author.

References should be placed at the end of the article and should conform to the style of the Quarterly Cumulative Index Medicus; viz., name of author, title of article and name of periodical with volume, pages, month and year. They should be numbered and should appear in the same order as that of the superior figures inserted in the appropriate positions in the text.

■ ILLUSTRATIONS—Illustrations accompanying manuscripts should be numbered and the author's name should appear on the margin or back.

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A reasonable number of halftone illustrations will be reproduced free of cost to the author, but special arrangements must be made with the editors for color plates, elaborate tables or extra illustrations. Copy for zinc cuts (such as pen drawings and charts) should be drawn and lettered in India ink or with black typewriter ribbon (carbon paper placed with carbon against back of sheet for bolder type). Ordinary blue or colors will not reproduce. Only good photographic prints or drawings should be supplied for halftone work.

■ CASE REPORTS—Case reports will constitute an important department of the journal and the editors welcome case reports that are of unusual clinical value or that present exceptional conditions of interest to dental surgeons.

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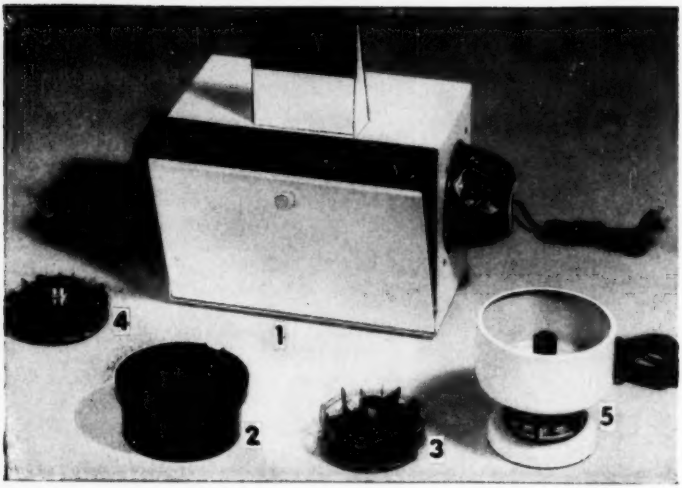

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